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An Address.[1]

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LET me first express to you my deep appreciation of the honour of addressing you on this occasion.

The year to come promises to be a critical one in the history of medicine in Australia. Decisions will have to be made of enormous consequence to the character and quality of medical services and to the status of the doctor in our community. Some of these decisions will be made by the public tomorrow at the polls, others we must make.

Last year the profession decided by a majority tantamount to unanimity to refuse to prescribe under the terms of the *Pharmaceutical Benefits Act*. In the year to come we may have to face one of two different situations. First, we may have to face the demands of a popular government for cooperation in a scheme of socialized medicine on dictated terms and conditions of which we have already expressed our grave disapproval. Alternatively, we must be prepared, as a profession, to put forward a sound and workmanlike scheme by which the best qualities, as we see them, in the medical services of today may be preserved, and the bad remedied. To do either we must be very clear on the principles involved. To this end I think there are two aspects of our problem which will bear examination, the first psychological, the second historical.

The psychological aspect is this. The arguments and discussions between profession and government have seemed to me to be touched with that almost Gilbertian unreality which occurs when two people, unbeknown to each other, are talking about two totally different things. The best illustration from our own point of view is the accusations which have been made against us in the course of the proceedings. The Prime Minister declared us to be an undemocratic body, coerced and hoodwinked into opposition by a dominating group of specialists who comprised the Federal Council.

A member of the House remarked that whenever he saw a child in agony with the toothache he felt like going out and shooting a doctor. Another said that he saw no need for privacy in medical affairs unless the patient had some disgraceful matter such as venereal disease to conceal. We were accused of being on strike. We were accused of deliberate persecution of the sick poor. I do not entirely believe that these remarks were directed as propaganda at the public. It is not improbable that the originators of the schemes were as puzzled as they were angry at our resistance and the public apathy which confronted their measures, as we were puzzled at their curious approach to the problem. The psychological difficulty, I would suggest, was that both parties were talking about totally different things, which they unfortunately called by the same name.

The historical factors that I would mention are of interest inasmuch as they help to shed light on the psychological difficulty as well as on the problem as a whole. Today, as our social institutions grow more and more complex, we must apply to the historian for answers to our questions. Even though it is stated that historians are invariably one-eyed, it is also true that in the country of the blind the one-eyed man is king.

The first historical principle is that a civilization or a community is a dynamic and not a static organization. The dynamic forces of history are perpetually at work. No community, as Toynbee points out, is ever emancipated

[1] Delivered at the annual meeting of the Queensland Branch of the British Medical Association on December 9, 1949.

from historical development. Those of us who, after both the 1914-1918 war and this last war, have found ourselves impatiently waiting for things to settle back to what we regard as normal merely betray our ingenuous lack of appreciation of this fact.

The next matter is that in all the civilizations of which we have record, and ours is only one of about 20 of whom 19 are dead or moribund, there has been a strong, even an inexorable, tendency for communities to divide into a small, wealthy, powerful and privileged minority and an underprivileged and subservient, generally exploited, majority. The original source and means of power in any instance may be military, religious, scientific (as in ancient Egypt), or economic, eventually it draws all other sources into its control. Much of history can be described in terms of the struggle for power, first the internal struggle between the classes within communities, and next that between communities for the various means of wealth and power.

In the western European civilization of which our community here is a colony, we have seen the power pass from the legions of Rome to the Christian Church, thence to the monarchical states, thence to the princes of industry and latterly to the voting majority of the population. The outstanding thing which has happened, however, is the enormous development of scientific knowledge and its application. This has led to the development of food production, manufacture, transport, communication of ideas, and public education on a scale unprecedented. The effect of this, for various reasons, has been that (to quote Toynbee) "the unequal distribution of this world's goods between a privileged minority and an underprivileged majority has been transformed from an unavoidable evil to an intolerable injustice".

Today, we as a profession are first of all the inheritors and trustees of the "technological know-how" as it relates to the health of individuals and the community. We live in a time, in the next instance, whose spirit is the extension as rights to every member of the community of such things as freedom of speech, education, personal liberty, and the like, which were formerly the privilege of the few.

Medically speaking, this extension of privilege has certain very curious aspects. The first is that it is technically impossible to limit the privileges of medical skill to any one class or section of the community. Public health measures must be nation-wide or fail. Similarly clinical medicine must be constantly maintained in its standards by the widest possible personal experience on the part of its practitioners. If a rich man maintains a personal physician for his own private service he must make a sorry bargain. It is for this technical reason as well, I hope, as for some genuinely humanitarian considerations that doctors have always staffed public hospitals for the poor willingly and diligently.

The next factor is that medicine demands for its efficiency violation of the privacy of the patient's life and affairs as well as of his physical body by the practitioner. We daily have to do things to patients which under any other circumstances would amount to abominable assault, and daily ask questions which under other circumstances would amount to abominable and degrading insult. And, moreover, we must carefully record and document our findings. It seems abundantly clear that the condition of professional secrecy which we share with our sister professions of the church and the law, and the responsibilities which go with it, must remain, whatever else happens, to the profession of medicine.

Here another factor enters. The historical facts as to the distribution of privilege and power within communities, together with other less creditable motives and less scientific generalizations than most historians are prepared to make, have led to the emergence of the idea of the supremacy of the rights of the State as opposed to those of the individual, that "the individual is nothing but a part of the society of which he is a member. The individual exists for society, not society for the individual. Therefore the most significant and important part of human life is not the spiritual development of souls but the social development of communities". I again quote Toynbee.

In the conflict between these last two sets of ideas, I would suggest, is the basis, the practical basis, of our opposition to the scheme of State medicine which we have been invited to support, and of our support of whatever schemes we may in the future advocate as wiser alternatives. A profession enlisted in a totalitarian State must degenerate technically into practitioners of a species of veterinary medicine. The only alternative to the technical aid of the privacy of the doctor-patient relationship is human experimentation on the lines of Dachau and Belsen.

What, then, do we want? Clearly the extension of the right of a private doctor-patient relationship to every member of the community regardless of financial status, and regardless of whether he is treated in hospital at public expense or otherwise. I personally cannot see that a State-controlled scheme of medicine, because of the necessities of policing public expenditure first, and second because of the ever-present threat of interference by theoretical politics, can ever have the whole confidence of patient or doctor. There are other equally good methods of removing the financial burden of sickness from the individual sick earner—these, I hope, we shall hear much of next year.

Finally, I would stress another point. In any dealings on professional matters we, as a profession, must speak with one voice. This is possible only if our minds are clear and our intentions honest. These I believe them to be. If that is so then it is of enormous importance that on matters of principle we stand firm. There can be no temporizing. I would remind you of Æsop's fable of the trees and the axe.

> A woodman went into the forest and begged of the trees the favour of a handle for his axe. The principal trees at once agreed to so modest a request and unhesitatingly gave him a young ash sapling out of which he fashioned the handle he desired. No sooner had he done so than he set to work to fell the noblest trees in the wood. When they saw the use to which he was putting the gift they cried: "Alas! Alas! We are ourselves to blame. The little we gave has cost us all: had we not sacrificed the rights of the ash, we might ourselves have stood for ages."

DYSMENORRHŒA AND THE CLIMACTERIC: PSYCHOSOMATIC ASSESSMENT AND TREATMENT.[1]

By A. A. MOON,
Sydney.

IT would require not only knowledge of the anatomy, physiology and pathology of menstruation, but knowledge of the whole woman, mind as well as body, before there could be full understanding of dysmenorrhœa and the climacteric. They have in common numerous subjective symptoms and a large psychic element, and no doubt the complex relationship between nervous system, endocrine system and reproductive organs is involved.

Dysmenorrhœa.

Primary (essential, intrinsic or spasmodic) dysmenorrhœa is excessive menstrual pain in the absence of any demonstrable pelvic lesion; secondary (acquired or extrinsic) dysmenorrhœa is always associated with disease or malformation of the pelvic organs. Before the subject of secondary dysmenorrhœa is dismissed, it is emphasized that alertness is necessary in diagnosing some of the less obvious developmental abnormalities, and that that ubiquitous disease, endometriosis, is to be kept in mind.

Primary dysmenorrhœa is mentioned in the Ebers Papyri (1500 B.C.) and was a clinical entity with recognized treatment at the time of Soranus (second century B.C.); yet today O'Donel Browne (1949) rightly states: "I do not know the cause." Matthews Duncan's definition in

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on November 10, 1949.

1879 of dysmenorrhœa as "a disease of the nature of neurosis in which the contractions of the uterus cause great pain" still covers our certain knowledge of the condition. The symptoms are well known and will not be detailed; but three important factors are to be considered in the assessment of dysmenorrhœa—psychogenic factors, endocrine factors and neuro-muscular factors.

Psychogenic causes are considered to be of outstanding importance by some authorities, who assert that correction of the psychic upset is followed by prompt cure. Novak and Harnik (1929) believe that every case of true uterine colic has its origin in psychic trauma; cure results when the original psychic trauma is brought to light and its role is explained to the patient. Dread of return of pain and fear of functional incapacity appear with succeeding attacks and produce dysmenorrhœa.

It is held, and not without reason, that equally good results follow all varieties of treatment—psychotherapy, general medical treatment, hormonal therapy and operative procedures. The nervous factor is considered so important that any treatment acting psychologically will yield a good percentage of cures, and it is stated that psychotherapy is the direct approach and gives the most effective and lasting results. Suggestion, either conscious or unconscious, is alleged to be of importance in treatment, and environmental and educational factors are stressed. Finally, primary dysmenorrhœa is considered by some authorities to be essentially a condition affecting the highly-strung, sensitive girl who has a lowered pain threshold.

The importance of psychoneurosis in the evaluation and treatment of primary dysmenorrhœa is not to be under-estimated, but it would not appear to be the primary cause or even the essential feature of the condition. We do not know the actual cause, and psychoneurosis is not to be used as a cloak for ignorance. Moreover, increased knowledge has explained some features hitherto considered psychogenic in origin. The pain of severe dysmenorrhœa is very real and would not appear to be psychic in nature; but the cumulative effect of cyclic pain inflicts mental trauma, lowers morale and sooner or later results in a psychoneurosis. Malingering is uncommon, although such a condition could readily serve the purpose of those who crave for sympathy or those who desire an escape defence.

Dysmenorrhœa is not limited to any social class. The hypogonadal type of woman—thin, highly strung and sometimes sexually under-developed—is often considered the dysmenorrhœic type. I find some of the most severe and intractable cases are among healthy country girls, well developed, free from conflicts or neuroses and otherwise quite normal with regard to menstrual history. Uterine hypoplasia is still mentioned in standard textbooks as a cause of primary dysmenorrhœa; but Jeffcoate (1945) and others have presented evidence suggesting that this view is unfounded. The warning is given that uterine hypoplasia is difficult to diagnose without actual measurement of the uterus with a sound, and the surgeon who readily diagnoses under-development of the uterus causes unnecessary worry to patients and may harm his reputation.

Dysmenorrhœa may commence at the menarche; but for another two to four years at least 60% of patients do not suffer pain of sufficient severity to warrant treatment. Are psychogenic causes exerting effects as unrestrained girlhood passes into the more difficult stage of womanhood? It is now recognized that there is a variable period of several years after the menarche when menstruation is, for the most part, anovular. It has been proved by Sturgis and Albright (1940) and others that primary dysmenorrhœa does not occur without ovulation. Marriage frequently cures or relieves the condition, and childbearing usually effects a cure. Is this a happy adjustment of earlier psychogenic problems, or is there some physical explanation? Clinically the uterus and the upper part of the vagina in many virginal women grow to their accredited normal size within a few months of married life if no contraceptives are used (Green-Armytage and co-workers, 1947). Normal intercourse would appear to have a beneficial effect on both the endocrine and neuro-circulatory mechanisms of the generative organs. The fear of pregnancy or practice of contraception can militate against this natural relief of dysmenorrhœa. The good results observed after childbirth and after dilatation of the cervix probably accounted for the theory of obstruction universally held in the days of Marion Sims. It is now known that cervical obstruction plays no essential part in ætiology. Bearing a child increases the tolerance of the uterus to stretch by enlarging it. The prolonged pressure of the presenting part on the cervix, and the effects of tearing on nerve fibres of and around the *cervix uteri* and *isthmus uteri*, probably eliminate an abnormal sensitivity of the nerve endings stimulated by strong and disordered myometrial contractions.

The similarity of pain in severe dysmenorrhœa to that of abortion or labour suggests abnormal muscle contraction as an important cause. Moir (1936) and others have shown that myometrial activity is most pronounced during the first and second days of the menstrual period, and the magnitude of muscle contraction is roughly in keeping with the degree of pain suffered. Frequent and irregular contractions of high amplitude associated with intra-uterine pressures of 120 to 200 millimetres of mercury are characteristic findings in patients with dysmenorrhœa. Moir considers that the uterine muscle is squeezed dry of blood during the acme of each contraction, and suggests that intrinsic dysmenorrhœa may be classed with diseases such as intermittent claudication and *angina pectoris*. Muscle is forced to contract in the face of an inadequate blood supply and characteristic pain results.

Spontaneous rhythmic contractions of the uterus can occur independently of the nervous system; but this does not imply, as Davis (1936) observes, that normal uterine contractions are uninfluenced by nervous impulses. Cotte and Davis consider that the presacral nerves show the changes of neuritis in severe dysmenorrhœa. The good results following presacral neurectomy and injection of the pelvic plexus point to innervation as an ætiological factor.

F. A. Maguire (1949) has recently made the following statement:

> All that we can say at present is that there are two separate nervous controls, sympathetic and parasympathetic; we do not know their distribution or the actual exciting influences. There is in addition a dual chemical control. Is the answer a nervous, chemical or embryological consideration?

Œstrogen-progestin balance is essential for menstruation, and hormonal control must be considered in the ætiology of dysmenorrhœa. Endocrine therapy has been popular; but as Hoffman (1945) remarks, "reported results have been inconstant, equally good results have seemed to follow opposite forms of treatment, and both good and bad effects have been described after identical therapy". Progesterone and gonadotropic hormones have been used to inhibit uterine contractions, but results of treatment have been conflicting and uncertain. Diseases such as *metropathia hæmorrhagica* are characterized by anovular menstruation, increased œstrogen levels and painless menstruation. Sturgis and Albright (1940), followed by Novak and others, prevented dysmenorrhœa by giving œstrogens in the first half of the menstrual cycle and suppressing ovulation, as proved by endometrial biopsies. This can be achieved conveniently by giving one milligramme doses of stilbœstrol daily for twenty days, commencing on the first day of the menstrual period. As a rule ovulation and pain recur after treatment ceases.

Androgens have been used on account of their depressing effects on uterine muscle, their inhibition of ovulation, and their ability to arrest menstruation temporarily. Doses of ten milligrammes of testosterone propionate twice a week throughout the cycle may be sufficient, and the dose should not be increased beyond a total of 150 milligrammes per month in case of undesirable masculinizing effects. Synthetic œstrogens usually yield better results than androgens, without the added risks and with less discomfort and cost to the patient.

Thyreoid therapy is indicated in the treatment of dysmenorrhœa when there is associated hypothyreoidism. Clinical signs of hypothyreoidism or a basal metabolic rate which is not elevated are the criteria for medication.

Much experimental work has been carried out in support of the theory of menstrual toxin and the presence of such a toxin is becoming recognized. George Van Smith (1947) considers that most of the phenomena of menstruation can be explained on the theory of menstrual toxin. The spasm of primary dysmenorrhœa is thought to be due to an exaggerated local effect of the toxin.

The roles of allergy, postural defects, insulin and vitamin *E* deficiency in the ætiology and treatment of primary dysmenorrhœa are not discussed in this paper. Certain general predisposing factors are of importance. Pain threshold studies by Haman (1944) showed that patients with dysmenorrhœa had the lowest pain thresholds of any group studied, and this low threshold was retained even after the climacteric. Clinically, patients with intractable dysmenorrhœa might have a lowered tolerance to pain, but this could be an effect rather than a cause of the disability. Constitutional, environmental and educational factors are important as secondary features contributing to lowered general health and morale. Herein lie possibilities for preventive and relieving measures. The education of girls at puberty is sadly neglected. All girls should be taught to regard menstruation as a normal function, which should not interfere with usual activities and habits. Mothers are frequently incapable of such education, and routine instruction by school medical officers would appear to be the method of choice.

The Operative Treatment of Primary Dysmenorrhœa.

Dilatation of the cervix is a time-honoured form of treatment which has been justified by results. It is indicated only when full medical treatment has failed. Cures are reported in from 25% to 50% of patients, and another 20% are relieved. Dilatation of the cervix is probably not so illogical as is sometimes thought, nor are the good effects based merely on strong suggestion. The operation can be considered a method of interrupting or altering the nervous mechanism closely related to the cervix and the isthmic segment of the uterus, and extending out to the ganglion of Frankenhauser. The practice of inserting a stem pessary after dilatation has lost favour and does not seem advantageous.

Injection of the plexus of Frankenhauser with one millilitre of 85% alcohol following dilatation of the cervix is said to enhance results and effect a cure in 60% of severe cases (Davis, 1936). The operation aims at causing degeneration of nerve tissue of the plexus, thus blocking impulses to and from the uterus at their nearest accessible site. The close relation of important structures such as the ureter, the rectal ampulla, and uterine and internal iliac vessels is to be remembered.

Presacral neurectomy has been widely used for the severe and intractable types of dysmenorrhœa since the work of Cotte in 1924. Advocates of this operation consider that the pain is chiefly neurogenic in nature, and the superior hypogastric plexus contains nearly all the sympathetic nerve supply of the uterus condensed in a narrow and accessible bundle. Results by many workers give a cure rate of 70% to 85%. The 15% failures are considered due to incomplete removal of nerve tissue owing to anatomical variations of the nerve components. Ingersoll (1946) advises a preliminary testing of patients with œstrogens to stop ovulation. If ovulation is inhibited and pain persists, the cause is considered essentially psychogenic and psychiatric treatment is preferred to surgery. O'Donel Browne (1949) considers that menstrual pain may be uterine, ovarian, or combined uterine and ovarian in origin. This differentiation can usually be made clinically, and a precise diagnosis is helpful before sympathectomy is performed. The uterine and ovarian nerve supplies are independent, and ovarian innervation is bilateral. Unless differentiation of the pain is accurate, Browne recommends the combined operation of presacral sympathectomy and bilateral ovarian denervation. Presacral neurectomy is a straightforward operation, the mortality rate is almost nil, and the immediate and late post-operative complications are no more than those of laparotomy. The operation should be performed only when all other measures to relieve the pain of dysmenorrhœa have failed. Assessment of this operation must take into consideration the lack of exact knowledge of the nervous control of the uterus, the knowledge available of the chemical control of the uterus, the 15% failures at operation and the risks of any abdominal operation.

Outmoded types of "tinkering" gynæcology in the form of douches and local applications have been abandoned as ineffectual. Low-dosage X-ray therapy and the intrauterine application of radium have also been abandoned as uncertain and unsafe. Hysterectomy now holds no place in the treatment of primary dysmenorrhœa.

Treatment of the acute attack of pain in dysmenorrhœa remains purely symptomatic, and is an unsatisfactory feature of practice. Rest in bed and the local application of heat are indicated when pain is severe. A variety of drugs—analgesics, antispasmodics, hypnotics, anti-allergens *et cetera*—have been used with varying results. Aspirin and phenacetin are the most popular drugs, and to these may be added such drugs as "Benzedrine Sulphate", codeine or propadine hydrochloride, according to prescribing art. Stronger analgesics such as "Nembudeine" and pethidine are used if necessary; but morphine and alcohol should never be given. Reports by Abarbanel and Rawlings (1949) show some relief of symptoms with magnesium gluconate treatment.

The Climacteric.

The climacteric marks the transition stage in a woman's life from maturity to senescence. Ovarian function fails, and with it there are loss of the child-bearing function, cessation of the menses and regressive changes in the reproductive organs. The majority of women pass through the "change of life" without symptoms causing interference with their general health. Severe symptoms are encountered in some 10% of women, and another 10% have symptoms of sufficient severity to warrant treatment.

It is to be remembered that the menopause is not generally a "difficult and critical time" in a woman's life. The normal, healthy woman reaches the climacteric not only with equanimity, but with a sense of thankfulness at release from her bonds. She has adjusted herself to psychological problems in the past and they now present no undue difficulty. She is freed from the discomfort of menstruation and from the fear of late pregnancy, and with due attention to the rules of living her general health, attractiveness and *libido* remain unimpaired for years. Nature has decreed that ovarian function and child-bearing shall cease at an age when ensuing years of good health and vitality can be enjoyed. Other glands, such as the anterior lobe of the pituitary, the thyreoid and the adrenals, are not senescent organs at this stage. Indeed, it is the cessation of this one member of the endocrine orchestra which is largely responsible for the disharmony which frequently occurs.

Other women have inherent weaknesses in their psychic make-up, which are prone to exacerbation at the climacteric as they are in convalescence and certain toxæmias. In the words of Greenhill (1946):

> If strong psychologic problems are associated with the menopause there is a suspicion that previous psychiatric disorder has existed. If a woman cannot adjust to the menopause this is only repetition of a pattern of difficult adjustment throughout life.

The climacteric may bring to such women an acute awareness of advancing years, in which difficulties associated with marriage and reproduction loom largely. Some fear the menopause as something unknown and not understood; others fear old age, lack of security and loss of attraction. The fears of late pregnancy, cancer, mental disease and heart disease are real problems. This background must be considered, and the first duty is to unearth each woman's problem and offer her some assistance in readjustment. Something more is required than the taking of a brief history, the performance of pelvic examination and a prescription for œstrogens. A careful history may be tedious, but is essential. The patient, in turn, responds with growing confidence in the medical attendant who seeks knowledge on the pattern of her life, her personality and temperament. This confidence is increased when a

detailed physical examination has been performed. Indeed, an orderly history and unburdening of troubles, in itself, does the menopausal patient a lot of good.

Ætiology.

Failure of the ovary to respond to the gonadotropic hormones of the anterior lobe of the pituitary gland is often preceded by decline of the *corpus luteum*, and anovular menstruation with abnormal vaginal bleeding is prone to occur. Finally the follicles fail to develop and œstrogen formation in the ovary ceases. There is a pronounced increase in the gonadotropic activity of the pituitary in keeping with the "see-saw" relationship between ovarian and pituitary function. Large amounts of gonadotropic hormones appear in the blood and urine, but it is not known whether the excess of gonadotropes or the initial œstrogen deficiency is the endocrine cause of the symptoms. Excess gonadotropic hormone may affect other endocrine glands, and possibly it influences the nervous system in some unknown way. An increased thyreotropic influence may result in the symptoms of hyperthyreoidism sometimes observed. Stimulation of the adrenal cortex and male hormone preponderance may explain certain masculinizing changes, and stimulation of the adrenal medulla with increased adrenaline output may account for many of the vasomotor disturbances of the climacteric. The occurrence of glycosuria and other evidence of disturbed carbohydrate metabolism suggests an altered pancreatic function. Many of the signs and symptoms of the menopause can be explained by such a theory; but knowledge is lacking on the effects of œstrogen deficiency or gonadotropic excess, or both, on the central nervous system, on the autonomic nervous system and on the ductless glands.

Symptoms and Signs.

Any abnormal symptom or sign occurring in women in their forties is prone to be ascribed to the "change of life". Ready acquiescence in such an outlook on the part of the medical attendant offers pitfalls in diagnosis. Novak (1948) stresses that an attempt should be made to distinguish those symptoms which are clearly menopausal in origin from symptoms frequently seen in women at the time of the menopause. Lack of harmony with surroundings, fear of the unknown and anxiety states in susceptible people can be followed by all the psychogenic symptoms commonly observed at the climacteric—irritability, fatigue, depression, headaches, insomnia, gastro-intestinal upset, vertigo, tinnitus, paræsthesia *et cetera*. These subjective symptoms occur in other groups of people, and the syndrome in soldiers is well known. Why should they be called menopausal symptoms because they happen to trouble a percentage of women at this epoch? Hot flushes are the most constant and characteristic symptom of the climacteric. Severe flushes may be followed by profuse sweats, and these symptoms may bring in their train weakness, loss of sleep, headache, tachycardia and dyspnœa. Vasomotor disturbances may also account for dizziness and headache by causing sudden fluctuations of blood pressure. These symptoms are directly due to endocrine disturbance, and hormone therapy gives good results. The "staircase" involution of menstrual function is associated with retrogressive changes in the body generally and in the reproductive organs in particular. Dermatoses and pruritus may appear, with atrophic and vascular alterations of the skin. Dyspareunia may follow atrophy and adhesive vaginitis. A further weakening of fascial supports may exaggerate an existing prolapse, with the production of characteristic symptoms. Atrophic changes in the urethra may cause frequency of micturition and dysuria.

Painful knee joints and tenderness above the condyles are the outstanding symptoms of a mild form of arthritis sometimes noticed at the climacteric. There is usually no swelling, no bone change and no limitation of movement. Opinions differ as to whether this is a true endocrine disturbance or traumatic arthritis due to weight-bearing effects on women who have rapidly gained weight.

Increased and widely fluctuating blood pressure readings are sometimes noticed at this epoch, and the condition is to be differentiated from the hypertension of cardio-vascular and renal disease. Climacteric hypertension would appear to be a manifestation of vasomotor effect due to endocrine disturbance, and œstrogen therapy frequently gives good results.

Diagnosis.

Diagnosis should not be difficult if the possibility of associated organic disease is remembered. Any discharge between menstrual periods, hæmorrhage between periods, increased or prolonged blood loss with menstrual periods and post-menopausal hæmorrhage are never normal. Malignant disease must be eliminated by recognized routine gynæcological investigation. Functional uterine bleeding, and other organic disease such as cardio-vascular disease, renal disease, psychoses *et cetera*, are to be kept in mind.

Treatment.

Much trouble could be avoided if suitable talks by competent people were given to women in their forties before the menopause began. Ignorance, misconceptions and fears bred of folk-lore and legend could be removed. A warning should be given to the patient that some discomfort might occur, but that there was no undue danger of serious physical or mental illness. The advantages of the menopause should be stressed, and a brief and simple explanation given of the nature of the change of life. A discussion on the patient's manner of life, personal traits and living habits will help in the task of mental adjustment. Advice should then be given on measures to improve the general health, and should include the correction of overwork, faulty diet, excesses in living, anæmia, obesity and constipation. Much has now been accomplished and many patients require no further treatment. Sedation may be necessary for those suffering from severe psychic disturbances. Phenobarbital is preferable to bromides or stronger drugs and should be given under supervision.

Psychotherapy, general medical measures and mild sedative treatment as outlined will cure some 80% of patients, and the remainder usually require endocrine therapy. Œstrogens are used as a means of tiding the patient over the stage of hormonal imbalance. Women who menstruate regularly although they are at or beyond the recognized menopausal age should not be given œstrogens. "The entire orderly process of sexual aging may be frustrated and the climacteric may be indefinitely postponed" (Hamblen, 1945). Œstrogens have carcinogenic effects on susceptible experimental animals, and it has been inferred that similar effects can occur in women. The doses given to experimental animals are, in proportion, many times greater than clinical doses, and there is no definite proof that œstrogen therapy can produce cancer in humans. It is agreed, however, that œstrogens should not be given to patients who have had previous treatment for carcinoma of the reproductive organs or breast. Œstrogens may cause uterine bleeding in women who have long since ceased to menstruate. Such bleeding is usually due to prolonged medication or to overdosage with the synthetic drugs, and requires investigation by diagnostic curettage. Gastro-intestinal symptoms are not common with menopausal doses of diethylstilbœstrol and can always be overcome by the administration of natural œstrogens. Œstrogens can become habit-forming drugs, and self-medication with proprietary preparations is common and is to be deplored.

Medication should be carried out according to a plan aiming at prompt control of symptoms, periods of interruption of treatment, reduction of dosage and cessation of treatment as soon as possible. A convenient routine method of treatment, after the manner of Greenhill, is as follows. A dose of 0·5 milligrame of stilbœstrol is given each night for twenty nights and omitted for ten nights; phenobarbital is given during the interval period if necessary. If results are satisfactory, the same dose is given every second day of the following twenty days' treatment and again treatment is suspended for ten days. A third course is given with reduced dosage and increased interval between doses, and the treatment is then stopped. If the original dose does not relieve symptoms promptly, the dose should be doubled and then progressively decreased.

Pellets or crystals of œstrogens are sometimes implanted in the subcutaneous tissues or beneath the vaginal mucosa for prolonged medication after the artificial menopause when stilbœstrol by mouth causes gastro-intestinal symptoms. Œstrogens administered in pessaries or ointments by the vaginal route are absorbed and are helpful in the treatment of adhesive vaginitis and kraurosis. Oral therapy is usually so effective that there is little need for other forms of treatment in the care of the menopausal patient.

The use of androgens for the control of menopausal symptoms would appear paradoxical, and they are usually not indicated on account of possible masculinizing effects, uncertainty in action and unnecessary expense. Androgens can be given when œstrogens fail to relieve symptoms, when œstrogens cause uterine bleeding, or when œstrogen treatment is contraindicated. Androgens depress the hyperactive pituitary gland and clinical results are said to justify their use. Doses of 10 to 25 milligrammes of testosterone propionate are given intramuscularly two or three times a week, not exceeding a total of 250 milligrammes a month. Combined œstrogen and androgen therapy has been used when œstrogens have been found ineffective.

Thyreoid therapy is of great help when there is associated hypothyreoidism. Dry thyreoid extract is given regularly over a considerable period under medical supervision.

Summary.

The evaluation of symptoms and treatment of dysmenorrhœa and the climacteric will remain difficult until more is known of the role of biochemical changes in psychoneurosis and more exact knowledge is available on the nervous and chemical control of the uterus. There remains a "no-man's land" between the pituitary, the hypothalamus and the cortex, and the link with the autonomic nervous system and the ductless glands is yet to be explained.

Many of the phenomena of primary dysmenorrhœa and the climacteric can be explained on a somatic basis. Perhaps increased knowledge of anatomy and biochemistry will result in less emphasis on psychogenic factors.

No attempt has been made to set out routine treatment, because each patient must be assessed psychologically, medically and gynæcologically. The gynæcologist has advantages over his colleagues in assessing such patients and has valuable medical and surgical therapy at his disposal. He cannot escape the task of evaluation of symptoms and must not confine his attention to the pelvis. He may with advantage attempt to apply some of the teaching of Grantly Dick Read: "To remove fear and induce a state of mind which is understanding and serene, and a state of body which is physically fit and muscularly relaxed and controlled." Recourse to expert psychiatric examination is usually unnecessary; but it is suggested that consultation with the psychiatrist should be obtained before a patient is submitted to a major surgical operation such as sympathectomy.

Excess medication, the too early resort to surgery, and over-emphasis on psychogenic causes are not in the best interests of the patient.

The organized medical education of girls at puberty, and women before the menopause commences, has been stressed. Women cannot be educated to seek treatment for early signs of cancer if normality in menstruation and the climacteric is not known.

In the words of Chamberlain (1947): "Gynæcologists and psychiatrists may, as diagnosticians, seem worlds apart . . . but as therapists they can aspire to be as one, united in a common objective to aid women."

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PSYCHOLOGICAL IMPLICATIONS OF DYSMENORRHŒA AND THE MENOPAUSE.[1]

By A. T. EDWARDS,
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THERE is a certain suspicion of Americanism in the title chosen for tonight's discussion—"psychological implications of dysmenorrhœa and the menopause"; it is reminiscent of those "best sellers", the psychology of salesmanship and the psychology of the fisherman. I feel that there is no place for such a title in psychiatry, for if we are to understand the symptoms of our patients, whether they are of the so-called psychosomatic type—peptic ulcer, hypertension and the like—or somatic manifestations of unresolved emotional conflicts such as headache, palpitation, enuresis or dyspepsia, or whether they produce symptoms that remain mainly in the mind—if we are to understand these symptoms, we must view them against the background of the individual as a whole. It follows, therefore, that it is most unlikely that a symptom such as dysmenorrhœa will have the same psychological background in all patients—in fact, every individual patient will present individual differences, though there exist definite trends in the personality make-up of patients who develop this symptom.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on November 10, 1949.

Dysmenorrhœa.

Dysmenorrhœa must rarely be the presenting symptom for which a patient is sent to a psychiatrist. Though to the psychiatrist are sent patients with varied physical disorders for personality evaluation, especially in hospital practice, I have not as yet been sent one patient with dysmenorrhœa. Patients of the neurotic type, who make much moan about their symptoms generally, rarely complain about dysmenorrhœa except in answer to a leading question. However, to such a question, a positive response is an extraordinarily frequent occurrence, especially if the question is directed towards the period roughly between the ages of sixteen and twenty-two years. This corresponds with the findings of Hunter and Rolf (1947), who considered that primary dysmenorrhœa began with the full establishment of sex consciousness and tended to end with the adoption of mature psychosexuality. This emphasis upon the personality factor appears frequently in recent authorities.

For instance, approaching the subject purely from the physical point of view, Florence Harding, advocating the administration of pregneninolen in this condition, found that her patients were much more neurotic than a similar number of women picked at random.

Kroger and Freed (1943) state that "a definite psychic factor exists . . . the principal feature is a lowered threshold to pain". Wittkower and Wilson (1940) believe that "personality factors are related to dysmenorrhœa in some way as yet undetermined". Hunter and Rolf (1947) are of the opinion that dysmenorrhœa is a "normal sensory conditioned process, reaching abnormal levels", and assert that all cases of even chronic secondary dysmenorrhœa have psychogenic factors in the background.

Fritz Wengraf (1944) regards dysmenorrhœa as an organ neurosis, showing the following characteristics typical of a so-called functional disorder: (i) There is an absence of any constant demonstrable abnormality. (ii) The symptom is inconstant, varying from month to month, mostly as a reaction to pleasant or to distressing events. (iii) It shows a connexion with other neurotic symptoms. (iv) It is amenable to most diverse modes of therapy, and frequently yields to suggestion or hypnosis.

One cannot agree fully with the opinion of Hunter and Rolf (1947), quoted above, as in many cases dysmenorrhœa commenced with marriage, followed the birth of a child, or followed introduction to sexual intercourse. It is rare to find dysmenorrhœa existing without other evidence of sexual maladjustment. The dysmenorrhœic is prone to fail to attain orgasm, or is able to attain only clitoric orgasm. This latter symptom, except in the case of those conditioned by long-continued and frequent clitoris masturbation, is an invariable indicator of psychosexual immaturity, almost always associated with a general failure of psychological maturation. These patients have a history of being spoiled by possessive, indulgent parents; they have never been weaned from these parents emotionally, and display unhealthy dependence upon them before marriage, and an abnormal seeking for their protection after marriage. As is usually the case, they seek the same indulgence in all interpersonal relationships that they found in their parents. They make "clinging-vine" demands from their lovers, whose ego is thereby flattered; but they continue to make the same demands for support and protection from their finally exasperated husbands.

Wittkower and Wilson (1940) found that their dysmenorrhœic patients were found with extraordinary frequency to have a history of maladjustment in childhood, and their patients generally had exhibited undesirable reactions to the onset of menstruation—disgust being the most frequent reaction.

This disgust typifies the general reaction of these patients in regard to the feminine role which they must play throughout life, and this role is denied in two contrasting ways, both being found in the dysmenorrhœic. The first—and in my experience the more common—method is that exhibited by the above-mentioned over-dependent (and on the surface typically softly feminine) individual, who is in reality denying her disgusting mature feminine self by clinging to reactions which represent the reactions of childhood in all important relationships with others, whether they are sexual or otherwise.

A not uncommon type of woman occupies the reverse side of the picture. Scorning her feminine sisters, she is generally the career woman, dressing to emphasize her scorn of the "pretties" of feminine attire, scorning sentiment, and assertive of the independence both of herself and of her sex. Her denial of her femininity is much more apparent, but she is much more likely to yield to it as the menopause approaches, with consequent reactions that frequently approach the psychotic. I doubt whether these women frequently seek help for their dysmenorrhœa; it is generally with extreme reluctance that they admit such a womanly weakness.

Overt homosexual behaviour or conscious homosexual feelings are common in these women, and it is with difficulty that they make an adequate heterosexual adjustment. The female role in sex relationships is perforce one of implied submission, and acceptance of male domination in any sphere being regarded as repugnant, domination in the very sphere that accentuates sex differences is excessively difficult to accept.

Most authorities agree in regarding dysmenorrhœa as being fundamentally due to a lowering of the threshold of pain as a result of personality difficulties such as I have mentioned. However, the associated symptoms would suggest that other mechanisms are also involved, in view of the associated neurotic symptoms that so frequently accompany dysmenorrhœa—premenstrual tension in both the psychic and somatic spheres, depression, headache of the neurotic type, spots before the eyes, "agonizing" backache, leg pains, mammary sensations, urinary and rectal disturbances, to mention a few. According to the individual conflicts—and especially when sex guilt is strong—such reactions may be the result either of conversion or of anxiety with its associated tensions.

Fritz Wengraf (1944) considers that the symptom is determined most frequently as the result of the first menstrual period, for which the patient is improperly prepared, becoming "the realization of old guilt feelings, evidence that the old apprehensions are becoming true and substantial. Most of these guilt feelings (have been) directed toward the mother, against whom the girl had exhibited aggressive, hostile or inimical thoughts." I have not been able to uncover the direct relationships between inimical impulses or thoughts directed toward the mother, and dysmenorrhœa, but I have no doubt of the truth of Wengraf's opinion, as I doubt whether any other mechanism is more frequent in the causation of other neurotic symptoms in the type of women most commonly subject to dysmenorrhœa. Freud's recognition of this fact led to his adoption of the now well-known Electra-complex—a half-truth.

Occasionally the conversion mechanism is refreshingly clear.

I recall two patients in their teens, both rather ignorant sexually, and both having been vehemently warned of the evils of masturbation by their respective mothers. Each of them suffered from dysmenorrhœa throughout the menstrual period, and each was able to relieve it completely each night by rubbing "the lower part of the stomach". Further investigation made it clear that the "terrible pains" were transformed sexual tension, that the *mons Veneris* had become an erogenous zone, and that rubbing the stomach was a euphemism for masturbatory activities resulting in nightly orgasm—not, however, recognized as such by the girls.

Adjuvant factors in the causation of this syndrome are ignorance and suggestion. Women still regard their catamenia with something approaching awe. They consider that they thereby rid themselves of evil humours and that a "missed period" is likely to send their blood to their heads with dire results—and as a consequence rationalize premenstrual headache as the result of delayed menstruation if their rhythm is of more than the accepted twenty-eight day interval. To the married woman not desirous of pregnancy, or afraid of it, each menstrual period is an ever-recurring threat, and one does not need very deep analysis to uncover this monthly anxiety state, with its consequent premenstrual tension. Popular literature now

abounds with cheap gynæcological as well as with pseudo-psychiatric advice and opinions. Articles in popular magazines for women are a constant threat to these patients, who read the articles therein with preconceived ideas, and extract from those articles only those parts that are in agreement with their ideas, with consequently increased introspection, further searching for symptoms, heightened anxiety, and the completion of the vicious circle of secondary anxiety that so often supervenes on primary neurotic elaborations.

In regard to treatment, I must leave to my colleague, Dr. Moon, any suggestions for drug or hormone treatment.

I have no personal experience of the use of hypnosis or suggestion in the treatment of these patients, such as has been used by Kroger and Freed (1943) with apparently promising results. It is likely to succeed mainly in the treatment of those patients who show evidence of being more than usually suggestible, and especially if there is evidence of other hysterical reactions.

Psychotherapy in these cases is not easy, especially for the more common immature type of patient. Treatment must be prolonged, and to be effective must involve what the psychoanalysts would call complete transference, with subsequent rebuilding of a more mature individual, especially in the psychosexual sphere. Only too frequently is this impossible, so that one is forced to endeavour to fit the environment to the patient, and to provide her with the necessary support and spoiling for which she craves.

Patients of the other type are more hopeful. They are more mature, and generally more intelligent, and are quick to gain intellectual insight into their problems. Emotional insight gradually follows, with satisfactory adjustment in an appreciable number of cases.

The Menopause.

The symptoms of the climacteric period are many and varied. On the somatic side are described hot flushes, sweating attacks, chills, palpitation, dyspnœa, vertigo, headache, paræsthesiæ, pruritus and hyperæsthesia. On the psychic side are emotional instability, depression, weeping, morbid worrying, insomnia, fatigue, self-depreciation, self-accusation, jealousy, suicidal thoughts. It may be stressed that none of these symptoms—and in fact no combination of these symptoms—is limited either to the age group or even to the sex involved. If self-depreciation and self-accusation are excluded, any permutations and combinations of the other symptoms are found in the neuroses, especially anxiety neurosis.

As Fritz Wengraf (1944) points out, menopausal symptoms to a great extent represent a continuation of patterns of reaction that generally appeared at puberty, and continued throughout adult life. These patterns of reaction are not only applied to menstruation, pregnancy and other manifestations of sex life, but tend to be reproduced in reactions to physical disorders of any nature as well as to the emotional stresses of life.

It is important to emphasize the fact that in most women the menopause occurs with no pronounced physical or emotional upset. This is a tribute to their underlying stability, in that they are able to resist the exaggerated and distorted disasters that gossip attributes to the mysterious "change", as well as to overcome the frequent suggestions of concomitant ill-health that the lay Press so often depicts as practically inescapable.

The adjustments required of a woman at the menopausal period of life are by no means only in regard to the physical changes that are occurring in her body. In a great number of cases there are required of her adaptations to changing conditions of life that are in no way directly related to these physical changes.

This period of life tends to coincide with the departure of the children from the home, and as in so many cases these children have represented the woman's sole real interest apart from the home itself, she remains without any buttress against introspection, with consequent consciousness of and elaboration of symptoms that would otherwise be disregarded. If in addition resentment and jealousy follow the marriage of her children, menopausal symptoms are an obvious means of once again strengthening the weakening bonds that her possessiveness had forged.

It is a well-known fact, of course, that much of our thinking occurs at the symbolic level, and there is no doubt that to most women menstruation symbolizes their femininity and therefore also their attractiveness to the male sex and to the husband in particular. Women approach the menopause, both natural and artificial, with trepidation, fearing the ensuing loss of sex attraction—and sex "is of man's life a thing apart, 'tis woman's whole existence". The artificial menopause as a result is often followed by temporary relative frigidity, even in those women whose sex life in marriage had been emotionally satisfactory.

Only too often these fears of losing her physical attraction for her husband are apparently justified by the fact that the menopausal period in the wife coincides with a lessening of sex potency in the husband. As a result he tends to seek relationships with his wife less frequently; or he, Faust-like, seeks a renewal of potency in the arms of some younger woman, where the novelty provides a temporary aphrodisiac. In either case the wife sees in her own "critical age" the cause of the husband's waning interest.

The dreaded changes now occurring in her sexual life tend to produce psychopathological changes at the menopause similar to the effect attributed by Wengraf to the onset of the first menstrual period.

Memories in the sexual sphere are reawakened, with renewal of the guilt feelings that were experienced at the time. One school of psychiatrists regards the emphasis given to these memories as purely fortuitous; but the weight of evidence points to the depression that so often attaches to these memories as being at least in part psychodynamically determined. Frequently the mental catharsis produced by full and repeated discussions of the problems here involved prevents the depressive mood from approaching the psychotic level.

Reinforcing the more specific mechanisms of the menopause is the fact that the climacteric period so often coincides with the vague—or clear—consciousness of deterioration that occurs around the completion of the fifth decade. In the male this consciousness of undesirable change is often rejected, with consequent depression or the development of neurotic rationalizations and elaboration. The woman invariably attributes it to the menopause, thus increasing her fixation upon the associated phenomena, and aggravating any existing emotional reactions.

Again I avoid suggesting physical methods of treatment. The literature of the endocrines abounds with so many conflicting results, in which the value of œstrogens, androgens and luteal substances is alike praised and condemned, that I leave the assessment of their value to the discussion which will follow.

For the more anxious or depressed type of patient, sedation is often necessary. I doubt if any one type of sedative has any particular value; in fact, it is generally advisable to change the sedative frequently, in order to avoid the patient's becoming over-dependent upon any one drug. I have a personal preference for a combination of "Sodium Amytal" and "Benzedrine Sulphate". I should like to stress the dangers of prolonged administration of large doses of bromide and long-acting barbiturates—a not uncommon cause of confusional psychosis.

For all patients with menopausal symptoms some exploration of the psychological background is advisable. As with dysmenorrhœa, so with the menopause, the first step is a simple—and repeated—explanation of the actual nature of the process. The naturalness of the condition should be repeatedly stressed, and frequent assurance should be given that the symptoms due to vasomotor dysfunction will gradually disappear as vasomotor control becomes adjusted. The patient should be first encouraged to relate her own conception of the menopause so that the usual misconceptions can be corrected. If possible, some discussion of situational problems should be carried out and an endeavour made to suggest some solution. The adoption by the patient of external interests to prevent introspection is frequently a *sine qua non*.

Depression occurring at this period must be carefully evaluated. If it is considered that it is probably part of a form of melancholia—manic-depressive or involutional—the risk of suicide must be kept constantly in mind. Fortunately, such depression responds satisfactorily to electro-convulsive therapy.

Finally, it must be emphasized that the recognition of psychodynamic factors in the ætiology of these conditions does not exclude the physical, but merely repeats the lessons to be learned from psychosomatic medicine—that every symptom, every disease, is not to be considered as a symptom or a disease existing in a patient, but is to be recognized as something influencing, or influenced by, that patient's total personality.

Summary.

Dysmenorrhœa and the menopause are both considered from the psychological point of view. The former is regarded mainly as an expression of psychosexual immaturity and guilt. The latter is influenced by various psychological phenomena associated with the period of life. Both are considered as expressions of the total personality of the patient.

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FACIAL PARALYSIS: A CLINICAL CLASSIFICATION.

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SINCE Bell (1830) described the lower motor neuron paralysis of the facial nerve all cases of paresis have been labelled "Bell's palsy", regardless of ætiology. Referring to this name, Kettel (1947) made the following statement: ". . . which far from designating an entity has become a collective diagnosis for all cases of peripheral facial paresis in which it has been impossible to demonstrate a local causation".

The history of decompression operations begins with the work of Ballance and Duel (1932). They considered the paresis as due to œdema of the facial nerve, which is compressed by an inelastic bony canal. Tickle (1948), Sullivan (1936), Kettel (1947), Cawthorne (1946) and many others have added to the work of the pioneers Ballance and Duel. During the last decade the operative technique has been advanced by Tickle, Sullivan, Kettel and Cawthorne. In 1948, while in America, I was fortunate enough to watch the work of Tickle (New York) and Sullivan (Toronto) on many occasions, also that of Kettel in Denmark.

In the section which discusses the causes of vascular occlusion facial paralysis, allergy is mentioned as a probable cause of the spasm or occlusion of the *vasa nervorum* of the facial nerve. More especially must we consider the vasoconstrictor or pressor substances, as pain is produced at the onset of the attacks; the pain is in direct relation to the anoxia resulting from the ischæmia. The explanation is simple: the larger the vessel affected, the greater the ischæmia and anoxia. The anoxic tissues produce a "*p*" substance. Here again is a relation of "*p*" substance to pain; this relation is demonstrated clinically by the history of pain given by the patient. It is suggested that the "*p*" substance is similar to histamine (vasodilator), but acts only on sensory nerves. This explains the initial pain; the continuing and occasionally increasing pain results from the increasing ischæmic œdema, which raises the tension in the inelastic Fallopian canal. Hilger (1949) states that "arteriolar spasm induces the ischæmic neuritis and œdema, especially where no collateral blood supply is to be found, as in the vertical part of the canal or where endarterioles are to be found".

I am not able to support a seasonal periodicity in the attacks, nor do I accept the cold theory (*a frigore*). In neuritic facial paralysis the ætiological focus is indicated as existing within the sensory field of the facial nerve.

The following classification designates the clinical entities of peripheral facial paralysis derived from the ætiological pathogenesis demonstrated at decompression operation, with a symptom syndrome from the criteria of each clinical group: (i) geniculate facial paralysis, (ii) neuritic facial paralysis, (iii) vascular occlusion facial paralysis, (iv) traumatic facial paralysis.

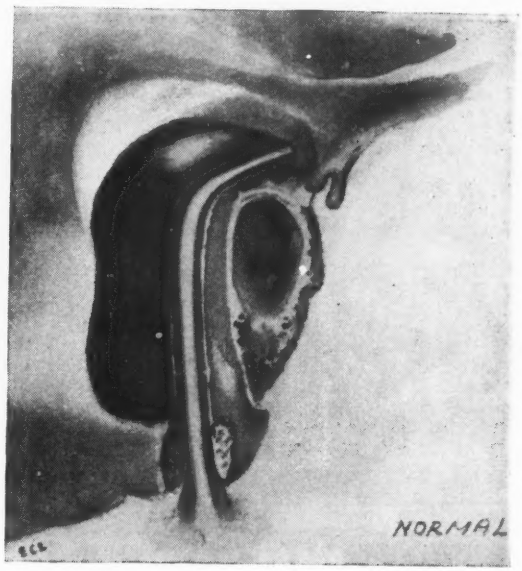

FIGURE I.

VARIATIONS OF ANATOMY OF THE FACIAL NERVE.

During operation and in the cadaver I have noted two distinct variations of the course of the facial nerve in the Fallopian canal, with changing position of the knee in its relation to the apex of the facial ridge. Figure I shows the normal position of the facial nerve. Figure II shows the first variation, which occurs frequently. This is a sharp downward curve or knee, commencing as the nerve passes into the interosseous partition of the medial wall of the aditus, just posterior to the incus, sweeping into the descending portion of the canal at the apex of the facial ridge, to continue a straight course to the stylo-mastoid foramen. Figure III shows the second variation, a wide bend beyond the postero-inferior border of the lateral canal, 3·0 to 4·0 millimetres posterior to the normal curve or knee. The descending portion passes across the mastoid process to the lower end of the facial ridge, about 3·0 millimetres above the stylo-mastoid foramen. At this point the nerve, having passed too far anteriorly into the facial ridge, kinks on itself backwards in order to pass out of the stylo-mastoid foramen.

I have observed this variation in only one case, at a decompression operation.

THE BLOOD SUPPLY OF THE FACIAL NERVE IN THE FALLOPIAN CANAL.

Three vessels are concerned with the arterial supply of the facial nerve through the *vasa nervorum*: (i) the

internal auditory artery, (ii) the superficial petrosal artery, (iii) the stylo-mastoid artery.

(i) The internal auditory artery enters the internal auditory meatus with the seventh and eighth nerves, supplying the facial nerve to the *hiatus facialis*.

(ii) The superficial petrosal artery, a branch of the middle meningeal artery, passes superficially along the petrous bone and enters the canal by the *hiatus facialis*; it supplies the facial nerve and anastomoses with the stylo-mastoid artery at the knee of the Fallopian canal, posterior to the tympanum.

(iii) The stylo-mastoid artery, a branch of the posterior auricular artery, passes over the outer surface of the facial nerve, ascends on the anterior surface, and enters the canal through the stylo-mastoid foramen, passing proximally in the canal to make an anastomosis with the superficial petrosal artery in the region of the knee of the Fallopian canal.

FIGURE II.

EXAMINATION.

History.

The most careful history is elicited from the patient, special regard being paid to the first signs or symptoms and their sequence prior to the appearance of the facial paralysis. Pain, malaise, headache, vomiting, giddiness, earache, tinnitus, deafness, sore tongue or throat and eye disturbance should be meticulously eliminated. If pain is the first and only complaint made by the patient I regard it as a cardinal symptom and sign, demanding a searching inquiry to establish the following: (i) the intensity of the pain; (ii) the duration, if the pain persisted after the appearance of the facial paralysis; (iii) the time relation to the sudden or gradual onset of the paralysis; (iv) the site of the pain, its localization or whether it is referred to any other part. The localization of the pain by the patient in giving his history is of the utmost clinical importance. To stress this fact I require the patient to pinpoint the spot in relation to the pain. If a tender spot or area is stated to exist by the patient, this becomes a major signpost, more especially if the patient localizes this painful spot behind the ear, over the Fallopian canal at the stylo-mastoid foramen or at the knee.

Routine Examination.

A thorough oto-rhino-laryngological examination is made for focal sepsis.

An ophthalmic examination is made: (i) the fundi are investigated for papilloedema; (ii) the corneal and light reflexes are tested; (iii) muscular function, especially that of the lateral rectus (sixth nerve), is tested.

Function of the facial muscles is tested by electrical reactions: (i) faradism and (ii) galvanism. For the purpose of clinical examination of this function I adhere to Kettel's practice (1947) of dividing the facial muscles into roughly three groups, corresponding to the divisions of the facial nerve: (i) superior, (ii) intermediate (middle) and (iii) inferior.

In 1939 Tickle wrote as follows: "In my experience the 85% that recovered had never lost their response to faradic stimulation . . . and 15% who lose their response to Faradism and show no improvement in six to eight weeks should have a decompression of the facial nerve."

My experience has been that 80% recover, and these patients had never lost their responses to faradism. As to the 20% that lost their responses in varying periods, as stated above, this was due to the grossness of the vessel lesion. Only one patient gave poor responses to galvanism, the cathode closing circuit; in some muscles the galvanic

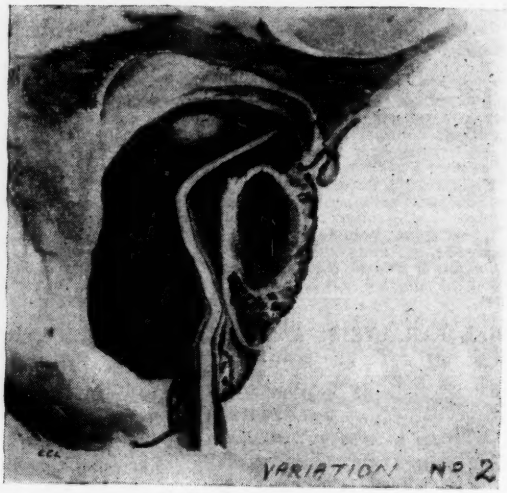

FIGURE III.

current required was as high as 10 milliamperes to evoke any response. The anode closing circuit gave greater contractions than the cathode closing circuit. The paralysis had persisted for ten months, giving complete reactions of degeneration. Following the operation, responses to faradism returned, and galvanic responses with the cathode closing circuit also returned to normal.

DIAGNOSIS.

Lesions of the geniculate ganglion produce facial paralysis, a cutaneous *herpes zoster* zone of the ear, an intraoral zone with herpetic eruption of the side of the uvula, soft palate, tonsil and fauces, and also *herpes zoster* vesicles on the side and dorsum of the anterior two-thirds of the tongue. Sensory disturbances may be present and localized to any area of the above distribution.

It is most important to bear in mind the possibility of a cerebello-pontine angle tumour. An examination of the ocular fundi is carried out; all eye reflexes are tested, especially the corneal reflex. I agree wholeheartedly with Phillips (1949) that a careful examination must be undertaken and if one is in doubt a lumbar puncture should be performed.

Pain is a most significant symptom of geniculate and vascular occlusion facial paralysis, and its intensity, localization and duration point to its ætiology.

The pain of geniculate involvement follows a period of malaise, and in all cases it is localized to the deep part of the ear, the patient always placing the tip of the index finger in the external canal, as if attempting to

obtain relief. The pain is described as neuralgic, not acute, lasting from one to fourteen days. The diagnosis is established with the appearance of the vesicles in the ear, on the palate and uvula and on the dorsum and side of the tongue, following an intense burning sensation. The paralysis is complete within forty-eight hours of the onset of the pain.

On the other hand, the pain of vascular occlusion is sudden, acute, severe and violent, and is localized behind the ear, either at the stylo-mastoid foramen or over the knee in the region of the mastoid antrum, persisting from twenty-four to seventy-two hours, although the duration varies within this interval. The facial paralysis is sudden and complete at the end of this period. The intensity and duration of the pain are intimately dependent on the size of the vessel suffering from the arterial block, whether the stylo-mastoid or superficial petrosal artery. The patient is well and suffers from no prodromal symptoms or signs prior to the lightning attack of pain.

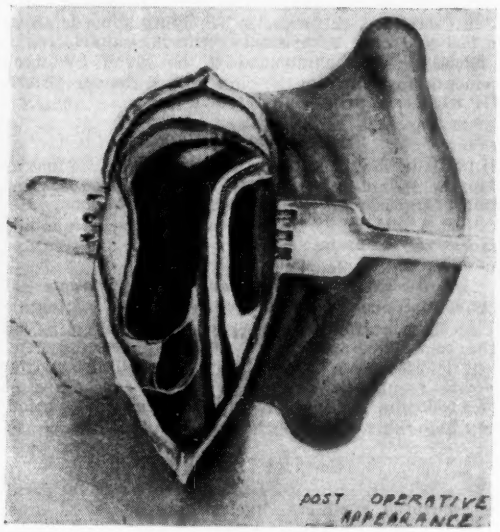

FIGURE IV.

In neuritic facial paralysis there is no pain and the paralysis develops slowly over two or three days and is always associated with focal sepsis or infection.

Traumatic facial paralysis presents no clinical difficulty in diagnosis.

GENICULATE FACIAL PARALYSIS.

Geniculate facial paralysis is a neurotrophic virus (*herpes zoster*) infection of the *ganglion geniculi* of the facial nerve, clinically manifested by an herpetic eruption of a cutaneous zone of the auricle, and of an intraoral zone involving the palate, fauces, uvula and tongue, associated with facial paralysis of the lower motor neuron type as described by Bell (1830).

Ætiology and Pathogenesis.

The causal agent is the virus of *herpes zoster* (a neurotrophic virus). *Herpes zoster*, mentioned by Landouzy (1884) as an infectious disease, is now recognized as a virus disease. The virus has been isolated by the use of the electron microscope (Boswell, 1947).

The majority of cases of *herpes zoster* are idiopathic or primary. The incubation period varies. The following periods are mentioned by Van Rooyen and Rhodes (1940): nine to twelve days, seven to fourteen days, and up to twenty-one days. These periods elapsed between the injection of zoster fluid and the development of the local vesicles.

The Portal of Entry of the Virus.

Van Rooyen and Rhodes state that "the portal of entry is unknown and various routes have been suggested". *Haslund (1900) states that "the entry may be through the* upper respiratory tract, nose, fauces, tonsils and then into the blood-stream and carried to one or more ganglia". Low (1919) states that the route is "from the nose by way of the olfactory perineural lymphatics to the meninges and *cerebro-spinal fluid, thence to the ganglia*".

Montgomery (1921) suggests that the virus enters the nerve endings in the skin and ascends along the nerve sheath in the lymph spaces until it reaches the appropriate ganglion. Montgomery considers that the paralysis is peripheral, the virus entering by the skin, although ascending in a sensory nerve it may attack a motor nerve causing paralysis. Van Rooyen and Rhodes support this view.

All patients suffering from the virus of *herpes zoster* examined by me have manifested the herpetic eruption within the geniculate zones of the sensory field of the facial nerve, and only those cases in which a full history and follow-up findings were available have been published. An analysis of the clinical signs and symptoms of the primary focal lesions points to the virus's having passed by way of the perineural lymph spaces of the nerves within the sensory field of the facial nerve to the appropriate ganglion or nerve.

The mode of entry of the virus, in my opinion, is through the mucous membrane of the mouth, fauces or tonsils, or via lymphoid tissue and from focal sepsis within the sensory field of the seventh nerve. This field includes the skin of the auricle. This portal of entry is consistent with the clinical histories of the six patients reported previously (Findlay, 1933, 1949). All had suffered from an attack of local focal sepsis within the designated sensory field of the seventh nerve, prior to the *herpes zoster* lesion of the geniculate ganglion, and the incubation period of the *herpes zoster* virus tallies with the lapse of time from the initial attack of focal sepsis to the outbreak of vesicles.

Van Rooyen and Rhodes make the following statement:

> The comparative frequency with which cases of Zoster Ophthalmicus are associated with paralysis of the eye muscles also lends support to Montgomery's theory, as he finds that it is the most superficial nerve, the oculo-motor, that is more commonly involved than the deeper trochlear or abducens. Other cases have been described by Roblin (1920), Rebattu (1933), Masser (1937) and Hunt (1915), who observed cephalic zoster associated with facial paralysis, superficial or Bell's type, thus pointing to the fact that the virus had attacked the nerve peripherally rather than centrally.

Symptomatology and Signs.

1. During the incubation period the patient may be pyrexial, and neuralgia may be experienced in the area which is soon to be affected (Van Rooyen and Rhodes).

2. I have found that most patients experience a burning sensation over the cutaneous zone to be involved, varying in duration from twenty-four to forty-eight hours. A patient with the intraoral zone and tongue affected complained bitterly of the burning pain prior to the appearance of the rash or vesicles.

3. Pain always precedes the palsy; the time relation is not constant. All patients suffer from this symptom in varying degrees. In *herpes oticus* the pain is deep-seated in the ear, ceasing generally with the cutaneous herpetic eruption. Duration of the pain varies from one to fourteen days. In the case of *herpes chorda tympani* the pain was localized in the tongue; no pain was experienced around the region of the ear at any time. Most patients place the onset of the palsy from twenty-four to forty-eight hours after the onset of pain.

4. Vertigo, vomiting, tinnitus and deafness will be present if the infective process spreads to the eighth nerve from the geniculate ganglion.

5. Herpetic inflammation of the geniculate ganglion is characterized by an herpetic eruption of the auricle and

intraoral geniculate zone (R. Hunt, 1915; Findlay, 1933, 1949). The vesicles may rupture with a watery discharge from the ear.

NEURITIC FACIAL PARALYSIS.

Neuritic facial paralysis is neuritis of the facial nerve originating from focal sepsis within its sensory field, characterized clinically by a facial paralysis without pain.

Ætiology and Pathogenesis.

1. Neuritic facial paralysis may arise by direct spread in the following ways: (i) by acute otogenous infection—(*a*) acute processes of the tympanum involving the epitympanic recess, (*b*) acute infections involving the tympanic antrum and the mastoid process; (ii) by chronic otogenous infection—(*a*) cholesteatoma of the epitympanic recess, (*b*) cholesteatoma of the antrum and mastoid process, (*c*) chronic suppurative lesions and osteitis of the bony framework of the middle ear cleft, especially the mastoid antrum and process. All the above-mentioned pathological processes may produce inflammatory neuritis of the facial sheath and nerve, the resultant œdema and compression causing strangulation, which slowly brings on the paresis.

2. Neuritic facial paralysis may arise by indirect spread by perineural lymphatics—(*a*) by bacteria, (*b*) by bacterial toxins (exotoxins). The indirect spread seems to me to be by way of the perineural lymphatics, the probable pathway of all chronic focal sepsis, the portal of entry by the infecting agents being from the local focal lesion with ascent to the distant appropriate ganglion and nerves. I agree with Montgomery (1921) in his thesis on virus infection:

> The neurotrophic virus enters the peripheral nerve endings in the skin and ascends along the nerve sheath in the lymph spaces until it reaches the appropriate ganglion.

When the perineural lymphatic spread is considered in relation to the distribution of the sensory field of the facial nerve, it means that bacteria and toxins may, from a septic focus in the mouth, teeth, tonsil, nose and postnasal space, enter the sheath of any nerve of this sensory field of the facial nerve and ascend to the appropriate ganglion and nerve.

To sum up the foregoing, the virus and toxic infection from focal sepsis seems to be governed by two main factors in both geniculate facial paralysis and neuritic facial paralysis:

1. The location of the septic focus determines the route which the causative agent will travel in the sensory field of the facial nerve to its final destination in ganglion or nerve.

2. The other determining factor points to the causative agent. A neurotropic virus is selective and will travel by the nerve route to the appropriate ganglion. The bacteria of chronic focal sepsis are generally low-grade organisms and their action and travel are sluggish, without much selectivity. This I think is emphasized in cases of neuritic facial paralysis, when the source and type of infection are considered.

Symptomatology.

The patient has only one complaint, and this is concerned with the loss of function of one side of the face. I have found in this clinical entity that psychic disturbances are prominent, and during treatment many patients become upset mentally, especially if the restoration of function lags. There is no pain whatsoever, and rarely are there any sensory disturbances prior to the appearance of the palsy. The facial paralysis creeps on them like a thief in the night.

An interesting feature of these cases is that no complaint is ever made about sore throats or abscessed teeth. In fact to the patients has come a shattering blow from somewhere which greatly upsets them. This state of mind is absent in those patients who have suffered pain; it has put them "on guard" and their reactions are reasonably normal.

THE TREATMENT OF GENICULATE FACIAL PARALYSIS AND NEURITIC FACIAL PARALYSIS.

The prime duty of the physician or surgeon is to allay the patient's fears, which are very real, the uppermost thought being: "Will I get better, and how long?" Throughout the treatment a cheery outlook must be maintained, with encouragement, and the slightest improvement must be commented upon.

Support of Sagging Muscles.

It is my firm belief that at all costs support must be given to sagging muscles at the commencement of the treatment. This may be achieved by using a plastic hook attached to the patient's first bicuspid or to the same position on an upper denture; it has no need to protrude beyond the red lip margin.

Massage.

Massage should be commenced at once and given at least three times a week. Only light massage is required, for two reasons: first, the muscles lack tone and may be injured; secondly, the muscles are lying superficially to a bone bed and may be bruised if the massage is too firm. All massage is recommended to be given by stroking movements upward from the chin toward the ear (Bierman, 1949), with extremely gentle touch.

Electrical Stimuli.

All patients should have galvanic massage, the maximum current being not more than three milliamperes. In my opinion faradism is contraindicated. Wilson, Kettel, Bierman *et alii* agree that the weak muscles should not be excessively stimulated.

Antibiotics, Chemotherapy and Vitamins.

I have not observed improvement in the patients who have received antibiotics, chemotherapy or vitamins, owing to the fact that lesions are well established when the patient is first examined. The new antibiotic aureomycin may give results in virus infections if there is a possibility of dosing the patient in the incubation period, or it may help when the rash is first manifested.

Surgical Treatment.

All my patients suffering from geniculate facial paralysis have recovered complete function of the facial muscles. It would appear that the virus infection of ganglia only produces an inflammatory reaction of the nerve in immediate close relationship; the resulting œdema is not gross. This is somewhat supported by the quick recoveries of function in five cases, as against one case in which over six months were required for restoration of function.

All my patients suffering from neuritic facial paralysis recovered clinical function with full mimicry in periods varying from three weeks onwards.

Therefore in geniculate and neuritic facial paralysis surgical decompression is not indicated, and these two groups constitute the 80% which make an uninterrupted recovery. On these grounds I endorse Tickle's (1947) figures, but emphasize the necessity of an accurate clinical diagnosis at the first examination of the patient.

Focal Sepsis.

The foregoing remarks in no way prejudice immediate surgical removal of local septic lesions in geniculate or neuritic facial paralysis, especially lesions of otogenous origin. Massage and electric stimulation are continued in hospital and during convalescence.

VASCULAR OCCLUSION FACIAL PARALYSIS.

Vascular occlusion facial paralysis is an occlusion of the *vasa nervorum* of the facial nerve in the Fallopian canal, characterized clinically by acute post-auricular pain associated with a sudden onset of complete facial paralysis.

Ætiology and Pathogenesis.

Two types of disorders of the *vasa nervorum* are responsible, allergic and vascular. The allergic factors are

(i) vasoconstrictor (pressor substances) and (ii) vasodilator (histamine). The vascular factors are (i) *endarteritis obliterans*, (ii) arteriosclerosis, (iii) embolism, and (iv) hæmorrhages into the facial nerve and sheath.

Before these factors are discussed it is necessary to appreciate that we are concerned with a vital structure confined in an inelastic bony medium, the Fallopian canal. Therefore, the loss of blood supply to the nerve becomes a "*Quellung* of the nerve" (Pollak, 1924), an œdema resulting in changes of degeneration of the nerve. This compression of the nerve in the canal causes further impairment of the vascular supply, giving rise to a secondary vicious circle. This is reversible, decompression relieving the compression and allowing the reestablishment of the blood supply to the nerve.

The Allergic Group.

Atkinson (1940, 1941, 1942, 1944) has published a great amount of work and has developed this ætiology for Ménière's disease, which, in my opinion, is analogous to the œdema of the facial nerve. Observations of the facial nerve *in vivo* during decompression have shown the œdematous areas at the stylo-mastoid foramen and at the knee of the canal.

In Ménière's disease Hallpike and Cairns (1938) noted at autopsy distension of the endolymphatic system with degenerative changes of the labyrinth. Unfortunately these patients are not examined till some days have elapsed, and whether intradermal histamine tests *et cetera* are indicated to establish allergy or whether they would materially aid the diagnosis and treatment is doubtful.

The sudden onset of the occlusion produces the ischæmia of the nerve which immediately gives rise to pain. The tension resulting from the increased œdema within the canal causes the pain to become violent, this again depending on the size of the vessel affected. Kettel (1947), summing up, makes the following statement:

> Much seems to indicate that Bell's Palsy is a pathogenetic entity, the primary and central feature of which is a "dysregulation" of the circulation, which probably takes place near the stylo-mastoid foramen. In most cases the nerve, as the most susceptible tissue, suffers alone. In other cases, the surrounding more resistant bone is affected. What the cause of this "dysregulation" is, and how the mechanism is released, I do not know.

In the occlusion of the vessel an ischæmic state of the nerve suddenly occurs, with subsequent paralysis. The experimental work on this point is discussed in a later paragraph. At this juncture it is necessary to recapitulate what I stated in the introduction relative to the anoxic condition of the nerve brought about by the ischæmia. The anoxia causes the nerve tissues to produce a "*p*" substance, the active agent in the production of the pain when sensory nerves are affected by a lesion of this nature.

I am prepared to accept the fact that the occlusion of the *vasa nervorum* of the nerve is due to pressor substances set free in the blood-stream. The sequence of abnormality producing the clinical symptom of sudden acute pain fits the picture of pressor substances, vasoconstrictor as against vasodilator bodies such as histamine. Undoubtedly the secondary œdema increases the pressure within the Fallopian canal, and so rising tension means more pain, and incidentally more damage to the nerve.

From my observations, sudden facial paralysis can occur only through a block to the vascular supply to the facial nerve, whether by allergic disturbance or by disorders of the vascular system. French authors Audibert, Mattei and Paganelli (1936) have propounded the following: "*La paralysie faciale périphérique dite 'a frigore' est fonction d'une atteinte artérielle des vasa nervorum.*" (Lower motor neuron paralysis is an arterial disturbance of the *vasa nervorum.*) As Kettel (1947) pointed out: "The term used is '*atteinte*' (slight injury) as against '*une lésion*', which would imply a serious prognosis and could not apply to the slight and transient type of paresis."

This sudden onset has not been noted in the neuritic facial paralysis group; most of the palsies were gradual, involving groups of muscles as the neuritic process spread. In the vascular occlusion group no sensory disturbances were noticed prior to observation of the paralysis by the patient.

I agree with Kettel (1947) that the paralysis of the nerve is due to the primary ischæmia; and the subsequently œdematous nerve is secondarily compressed in the Fallopian canal. Considerable support is given by the investigation and work of Lewis, Pickering and Rothschild (1933), who studied the paralysis induced by applying pressure to human limbs with the sphygmomanometer cuff; by applying localized pressure on single nerves they showed that the paralysis was not due to pressure on the nerves, but to local ischæmia. Other investigators, Denny Brown and Brenner (1944), have stated the same opinion: "The effect of pressure on the nerve is considered to be due entirely to ischæmia."

The term "occlusion" is used and adhered to in this allergic group instead of "spasm". The explanation for this is to be found in the clinical symptom of severe pain, which in one case persisted for seven days, and in the obviousness of the lesions demonstrated in the sheath and facial nerve at decompression operation. It is within reason to suppose that more than one vessel of the *vasa nervorum* may be involved in the occlusion or that the block may occur in one of the end branches of the artery supplying the specific region where the lesion is noted. Wolf and Wolf (1948) agree that pain arises from lesions of blood vessels owing to their sensory nerve supply.

The Vascular Group.

Wilson (1940) commented that "vascular disorders within nerve trunks provide another set of causes via the *vasa nervorum*". I have been able to satisfy myself from the examination of the sheath and nerve *in vivo* that many of the above vascular disorders were present. No microscopical investigation has been possible, as no subjects have come to autopsy.

Cawthorne (1946), using a dissecting microscope, noted hæmorrhagic streaks at the stylo-mastoid foramen. Many others, including Morris (1938), Collier (1940) and Horgan (1939), have noted œdema of the nerve. It would be possible to detect evidence of hæmorrhage macroscopically. Dilated vessels and hæmorrhagic streaks of the sheath and nerve are easier to observe when a dissecting microscope is used; "6×" magnification gives a very good picture; "10×" magnification is required for a detailed spot.

I have noted staining and dilated vessels at the knee of the canal at decompression in one case, and in another case dilated vessels and streaks in a very œdematous sheath, with considerable staining of the nerve at the stylo-mastoid foramen. The œdema of the sheath was so severe that a well-defined constriction of the sheath showed the impression of the foramen.

Symptoms.

The chief symptom complained of by patients is pain. The pain is sudden and severe and may be violent and acute, attacking without any warning patients who are in good health and pursuing their daily tasks. There is some variation in time from the onset to the appearance of the palsy. In my cases it has been from twenty-four to seventy-two hours. The pain may persist for some time after the palsy has been noticed.

The pain is always localized behind the ear, on the mastoid process in one or other of two positions, and the patient can, when asked, pinpoint the spot of maximum intensity. The spot is always tender to touch. The first spot is over the tip of the process slightly towards the anterior border. This would correspond to the surface marking of the stylo-mastoid foramen. The second spot corresponds with the lower border of the mastoid antrum, thus designating the knee or bend of the nerve in the medial wall of the antrum. The pain does not radiate, but gradually eases. The facial paralysis is sudden and complete, occurring at the end of twenty-four to seventy-two hours. Psychic disturbances have not been observed in cases of this group, and I have found most patients cooperative and helpful.

Surgical Treatment.

In the performance of the decompression operation on the facial nerve and incision of the sheath in the Fallopian canal two avenues of approach are available: (i) the endaural incision with the attico-antrotomy route, (ii) the posterior auricular incision. Either of these methods gives a good field of the mastoid process and tympanum.

The operation now performed by me is a combination of the above approaches, following a posterior auricular incision. This technique was evolved from my personal observation in 1948 of the operative technique of Tickle (New York), Sullivan (Toronto), Kettel (Denmark), Passe (London), Simpson Hall (Edinburgh) and Cawthorne (London). As the result of experience gained from many dissections of the Fallopian canal and facial nerve on the cadaver, and from early operative work (reported in 1946), the posterior auricular incision is advised, as it gives a free approach to the mastoid tip, especially the anterior part.

TRAUMATIC FACIAL PARALYSIS.

Ætiology and Pathogenesis.

Traumatic facial paralysis may be produced (i) by accidents—(*a*) fractures of the base of the skull or (*b*) fractures involving the mastoid process, or (ii) by operations — (*a*) simple or radical mastoidectomy or (*b*) fenestration.

I reported a case of this group in 1946, in which good mimicry was recovered following decompression six months after a fracture of the base of the skull. In another case of a fracture of the base of the skull the occipital bone, the petrous temporal bone and the mastoid process were involved, and the fracture passed through the knee of the Fallopian canal and continued into the apex of the facial ridge. At a decompression operation after the accident the facial nerve was œdematous at the knee and dilated vessels were noted in the descending portion.

Surgical Treatment.

Surgical treatment is (i) by decompression, (ii) by nerve graft, (iii) by end-to-end anastomosis of the facial nerve.

PROGNOSIS.

Electrical tests may not help in the forming of an opinion about prognosis. Kettel (1947) expresses this view:

> When muscles are paralysed their excitability to electric stimuli is increased, the chronaxia is shortened, after a short period the excitability of the muscle decreases; therefore, the chronaxia is lengthened. . . . The negative Faradic reaction by no means excludes a complete spontaneous restitution.

It is my intention to consider the prognosis of each clinical entity individually.

Geniculate Facial Paralysis.

The outlook is most favourable for a complete recovery of function of all facial muscles within two months, the longest interval in my experience being twelve months. I am not able to state definitely whether an attack by a virus confers immunity. Of my series one patient only had two attacks, an interval of twelve months separating the lesions. In all cases of geniculate facial paralysis aggravated by focal sepsis immediate surgical removal of the foci is indicated. Failing this, a guarded opinion as to the return of muscular function is given.

Neuritic Facial Paralysis.

In my opinion patients with neuritic facial paralysis all do extremely well, the prognosis depending firstly on the time factor—how long the paresis existed before the patient sought advice and treatment—and secondly on the location and extent of the focal sepsis detected at the first examination and how soon it was extirpated. The immediate removal of focal sepsis ends the bacterial and toxin invasion, and I have observed clinical improvement of muscular function within three weeks.

The question arises in these types of cases, when to advise a decompression of the nerve if delay in clinical improvement persists. I agree with Kettel that after a period of six to eight weeks has elapsed without some clinical improvement operation is advisable. Again, in this group some patients improve in condition and maintain the gain but then cease to go ahead. I advise operation and recommend that the entire length of the Fallopian canal be decompressed from the geniculate ganglion to the stylo-mastoid foramen; it may be necessary to sacrifice the incus.

As was stated previously, patients in this clinical group and in the geniculate group are the 80% who recover the lost function.

Vascular Occlusion Facial Paralysis.

The prognosis in vascular occlusion facial paralysis is good, provided a diagnosis has been made at the first examination and early operation is advised. There is no clinical reason to delay the decompression of the nerve. The symptoms are clear-cut, pointing to an arterial block. Therefore it is imperative to relieve the strangulation and promote a blood supply to the nerve. I do not agree with Kettel (1947) that operation should be held in abeyance for six weeks; this permits irreparable damage to the nerve. I support his statements that patients who were operated on early seemed to lag in showing any clinical improvement; but in my opinion those cases quoted by him belong clinically to the neuritic facial paralysis group. When his clinical histories are analysed few of his patients gave a history of pain situated behind the ear or mentioned the quality of the pain. I noted with interest that in those cases in which pain behind the ear was present macroscopic changes of the sheath, nerve and bony canal wall were found on decompression. This supports my contention and operative findings that the lesion is due to an arterial block, and to obtain a good result an early decompression operation on the facial nerve is indicated. Again, I do not agree with Kettel that all patients with facial paralysis suffer a "dysregulation of the vascular supply", and I have endeavoured to answer this statement by postulating the four clinical entities.

It is my firm belief that these patients will not recover full mimicry or restoration of complete function of the muscles unless a decompression operation on the facial nerve and incision of the facial sheath are carried out as soon as possible. Horgan (1939) performed a decompression operation fourteen days after the onset of the paralysis, with an excellent result. He noted pronounced œdema and bulging of the nerve.

Traumatic Facial Paralysis.

In traumatic facial paralysis a guarded prognosis is given, especially in those cases in which a graft is to be used, as function may not return for a considerable period—from six to fourteen months. In cases of fracture of the base of the skull a favourable prognosis may be given provided decompression is carried out as soon as the patient is considered to be in a fit state for the operation.

In my opinion the decompression operation should not be contemplated until six weeks have elapsed, for the following reasons: (i) to permit fibrosis of the blood clot in and around the fracture lines; (ii) to observe whether there are any signs of clinical restoration of function in the nerve; (iii) to allow firm healing to seal off the operative field from the possibility of intracranial infection.

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THE VALUE OF THE PROTEIN HYDROLYSATE CAPAIN ("AMPARON") IN THE TREATMENT OF TRAUMATIC SHOCK.

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THE pathogenetic factors of traumatic shock are not yet fully understood. Abnormal permeability of the walls of the capillaries has for many years been considered to be one of the fundamental causes of the failure of circulation in secondary shock. Therapy, largely based on this principle, resulted in great successes and up to the present the intravenous administration of blood and plasma is therefore considered to be of primary importance.

Recent studies revealed, however, that hæmoconcentration and loss of plasma through abnormal leakage of capillary walls are by no means the most frequent causal factors of the circulatory disturbance in traumatic shock (Mills and Gordon, 1946). More and more attention is paid to the chemical side of the question. Corelli (1946) holds that disturbances in mineral metabolism play an important role in shock conditions; Gregersen (1946) points to the consequences of acidosis. Histamine and allied substances are coming more and more to the foreground, and thus the original conception of traumatic shock, modernized and extended, as an acute alteration in the autonomic nervous system with its cerebral (and especially hypothalamic) connexions, concerning its cholinergic as well as its adrenergic and its histaminergic functions, plays its role again.

The study of the various ætiological factors in traumatic shock remains a difficult one: first, because of the urgency of affairs, which does not allow experiments; secondly, because of the fact that, in view of the universal acceptance of the superiority of blood and plasma in shock treatment, it would generally be considered a serious mistake to delay their administration. The successes obtained with plasma and blood transfusions may lead to the opinion that shock therapy has reached its goal and that the only problem left is, eventually, to find suitable substitutes for plasma and blood, as the supply of these is bound to remain limited.

But are we justified in accepting as an established fact the superiority of plasma and blood in the treatment of traumatic shock? What about the failures of this therapy that actually occur? Is it correct to attribute them, when morphologically demonstrable causes of death are lacking, to so-called "irreparable shock damage already inflicted to vital systems" before the above-mentioned "supreme" therapy was possible? We know that several investigators are convinced that even blood transfusion therapy will fail in those cases in which shock conditions with severe hypotension—or even a blood pressure only slightly subnormal because of compensatory vasoconstriction—have lasted too long. Besides, it should be borne in mind that apart from derangements of a physical nature, disturbances in biological functions, irregularities in biochemical processes, which often are detectable only by special laboratory investigations, may equally lead to a fatal outcome when not combated efficiently. Are we sure that plasma and blood are equally supreme in combating these biochemical disturbances directly, or are we to explain their undeniable course will have its favourable effects on biological effect thus, that "by giving the supreme plasma (blood) therapy, circulation as such will recover, and this of course will have its favourable effects on biological functions *et cetera*"? It will be clear that at any rate this therapy is rather symptomatic.

It happened, partly by coincidence, partly under the stress of war circumstances, that about the end of 1943 experiments were started with a casein hydrolysate, "capain", prepared by Professor R. Brinkman at Groningen, Holland. It consists largely of polypeptides (averaging octopeptides) side by side with a fraction of free amino acids. It is obtained by the hydrolysing action of the vegetable ferment papain on casein, and possesses a considerable water-attracting power. About this and other

properties of capain a preliminary report has already been published (Brinkman, Eerland, Hissink and Vegter, 1945).

The clinical trial was based on the water-attracting properties of capain, with the idea of thus fixing a sufficient volume of circulating fluid in the blood vessels, the oncotic attraction of a 13% solution of capain being about seven times that of plasma. It exerts a considerable osmotic pressure (more than two metres of water) against plasma and maintains it for ten hours or more. It was only afterwards that the vasoconstrictor properties of capain were brought to light by careful observation of our first patients; they were then confirmed by animal experiments.

Before capain solutions were tried in the treatment of shock it had to be ascertained that capain molecules, being smaller than native albumin molecules in blood, did not "leak" too quickly through "permeable" capillary walls, with possible dangerous effects by binding water extravascularly. And what did we know about the speed of assimilation of these polypeptides and amino acids in a shock-damaged organism?

Careful experiments were started to ascertain whether capain could maintain its water-attracting (water-binding) properties after intravenous administration long enough to draw circulation out of the vicious circle into which it had been brought by the shock-causing trauma. The fate of the capain molecule was studied after intravenous injection in patients suffering from serious hypoproteinæmia with œdema. In these cases we found that capain remained in the blood-stream for about five to six hours. The condition of the capillary walls in serious hypoproteinæmia, however, cannot be considered as the analogue of the condition of the "permeable" capillary walls in traumatic shock.

In view of the lowered metabolism in shock it seemed probable that the assimilation of capain polypeptides in protein metabolism would be retarded too. Therefore, the possibility of "capain intoxication" by overflooding the organism with amino-acid materials seemed worth consideration.

The working scheme in our clinical experiments with capain was as follows.

1. The shock patient was to be got out of the circulatory disturbance as quickly as possible by a rapid intravenous injection of concentrated solutions of capain, in order to give its water-attracting and water-binding qualities the best chance to develop intravascularly. If this could be realized, it was hoped that circulation would recover in a sufficiently short time, that is, within the period of intravascular localization of the capain molecules.

2. As soon as a sufficient amount of capain had been introduced in this quick way its administration was to be continued by slow intravenous infusion of more dilute solutions in physiological saline or 5% glucose solution.

3. After definite recovery of circulation capain would no more be needed as a water-attracting and water-binding agent. Then the recovered metabolic processes might use it in the synthesis of native proteins. Thus the patient, only just recovered from his shock by the "anti-shock" qualities of capain, might make good use of the nutritive properties of the same preparation. The fast injection method, however, can be performed only under strict supervision of heart, lungs, circulation *et cetera*.

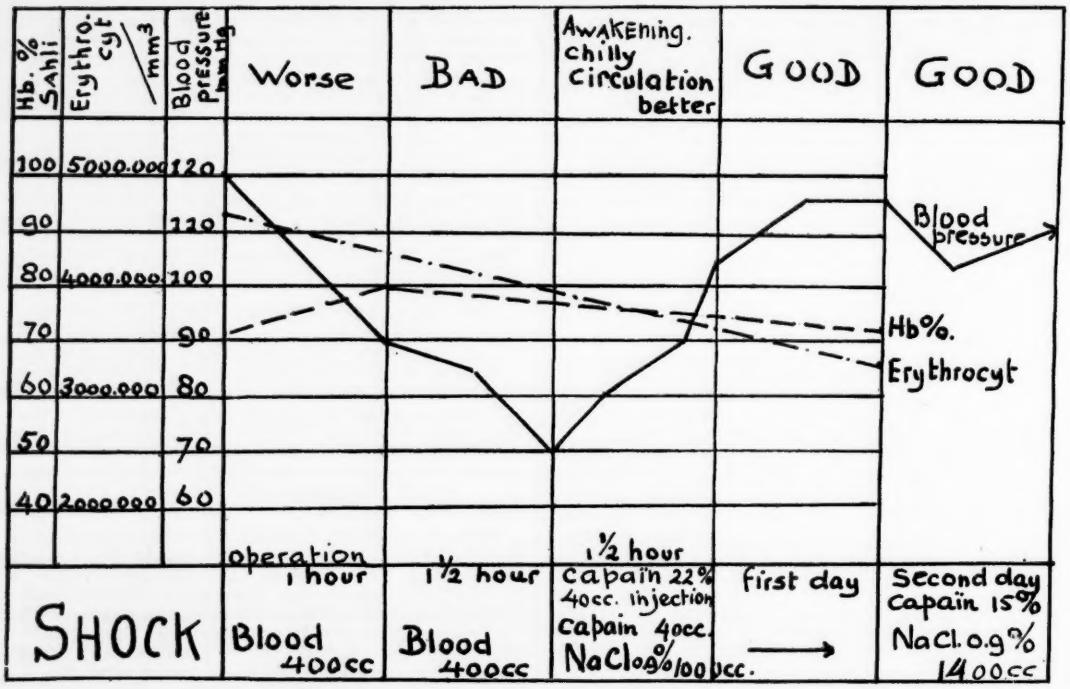

FIGURE I. Case VI.

Case Reports.

Out of 67 cases five are reported in detail, as follows.

CASE VI.—A woman, aged twenty-one years, with a thoracic empyema (non-specific) of long standing, which had been treated elsewhere partly conservatively and partly by drainage methods without favourable results, was admitted to our clinic to be operated upon by thoracoplasty. Her condition was moderate, her blood pressure was 120 millimetres of mercury (systolic) and 70 millimetres (diastolic), and her hæmoglobin value was 70% (Sahli). Undoubtedly she had hypoproteinæmia. A complete thoracoplasty was performed (after Scheede); during operation, which took one hour, a transfusion of 400 millilitres of whole blood was given. At the end of the operation the condition was bad and another blood transfusion of 400 millilitres was given at low speed. Shock developed, the blood pressure falling

to 90 millimetres of mercury (systolic). In the following hour and a half her condition grew worse and the systolic blood pressure measured 70 milligrammes of mercury only.

A quick intravenous injection of 40 millilitres of 22% capain solution was then given with the immediate result that the patient awakened out of her coma. Circulation improved visibly. The injection was followed by slow infusion of a more diluted capain solution (40 millilitres of 22% capain solution in one litre of 0·9% saline solution). This infusion took about one and a half hours. A chilly reaction of fifteen minutes' duration was observed, without unfavourable effect; on the contrary, circulation recovered quickly, and after this medication the condition was good and blood pressure measured 110 millimetres of mercury (systolic) and 70 millimetres (diastolic). Recovery was further uneventful.

Summary.—This patient with hypoproteinæmia certainly was predisposed to develop shock after a great surgical trauma. Transfusion of 800 millilitres of blood at low speed failed to restore circulation; on the contrary, shock deepened seriously. The quick injection of concentrated capain solution immediately had favourable effects by awakening the patient and raising the low blood pressure. It is probable that the vasoconstrictor activities of capain played an important role in this recovery. In this case a dangerous primary shock component existed, which was not efficiently combated by the transfusion of blood. By its continued disastrous effects on circulation the fatal outcome seemed certain. Yet capain brought recovery.

CASE XLI.—Operated upon in 1932 by open drainage of a thoracic empyema, a boy, aged twelve years, had a fistula in the left axilla and a remaining empyema in the thoracic cavity with many adhesions. The boy's condition was bad. On September 7, 1944, a thoracoplasty was performed, in which the first four ribs were resected subperiosteally. During operation physiological saline and blood were given intravenously (1000 and 200 millilitres respectively) in one hour, but after operation a serious condition of shock was evident. Another blood transfusion of 300 millilitres was given in half an hour's time, but the condition remained bad, with a blood pressure of about 90 millimetres of mercury (systolic). Infusion was continued by injecting one litre of 0·9% saline solution combined with two millilitres of paredrinol ("Veritol"). The pulse rate was 130 per minute. This amount of fluid was infused in two and a half hours' time, but the result was disappointing: the condition remained bad and the systolic blood pressure stayed on the same level of 90 millimetres of mercury. Then blood was given anew (400 millilitres in half an hour) and the systolic blood pressure rose to 95 millimetres of mercury, but the condition did not improve; so infusion was continued with one litre of 0·9% saline solution. The systolic blood pressure dropped to 90 millimetres of mercury, and the condition seemed to be growing worse. Then 50 millilitres of 15% capain solution, diluted with 500 millilitres of 0·9% saline solution, were infused very slowly (in about three hours): the blood pressure rose immediately and general recovery was evident. The patient recovered uneventfully.

Summary.—In this case the hypoproteinæmic patient did not recover from his condition of post-operative shock despite several medications, and in spite of a large quantity of blood that, however, had been infused too slowly, as was the case with the other infusions (physiological saline and saline combined with paredrinol ("Veritol")). It seems probable that faster infusion might have had more favourable results. Capain, although also infused very slowly, was more efficient, probably owing to its vasoconstrictor activity combined with its water-binding power. The patient recovered quickly and definitively.

CASE IV.—A woman, aged thirty-four years, was admitted to hospital to be operated upon for persistent jaundice. Her gall-bladder had been removed three years before, but soon jaundice developed. One year later she was operated on again, yet jaundice did not subside. At examination the patient was found to have a large solid liver and spleen, so that a diagnosis of cirrhotic liver was made. Obstruction of the common duct was considered as a possible cause. Her condition was bad: the blood pressure was 110 millimetres of mercury (systolic) and 75 millimetres (diastolic), the pulse rate was 70 per minute, and the blood urea level 375 milligrammes *per centum*. On January 20, 1944, laparotomy was performed, and a rubber prosthesis was put in place. Much diffuse bleeding occurred. The liver showed cirrhotic degeneration. After operation the patient was in a serious state of shock: the blood pressure measured only 85 millimetres of mercury (systolic) and 65 millimetres (diastolic), the hæmoglobin value was 64%, and the erythrocytes numbered 3,680,000 per cubic millimetre. A large blood transfusion was given: about 1200 millilitres went in, in about one hour and a half. Her condition, however, worsened dangerously and the blood pressure measured only 50 millimetres of mercury (systolic) after the transfusion. To the remaining blood (100 millilitres) 50 millilitres of 22% capain solution were added, and from this very moment onward the condition improved visibly. The blood pressure rose slowly, and infusion was continued by giving 20 millilitres of 22% capain in 100 millilitres of 5% glucose solution; this went in, in half an hour, with the same favourable result. Then 200 millilitres of 5% glucose solution were infused without addition of the water-binding capain. The patient's condition maintained itself, and the blood pressure continued to show a trend to rise. In the next three hours 600 millilitres of glucose solution together with 20 millilitres of 22% capain solution were given. This therapy was continued by infusion of 500 millilitres of saline to which 20 millilitres of 22% capain solution had been

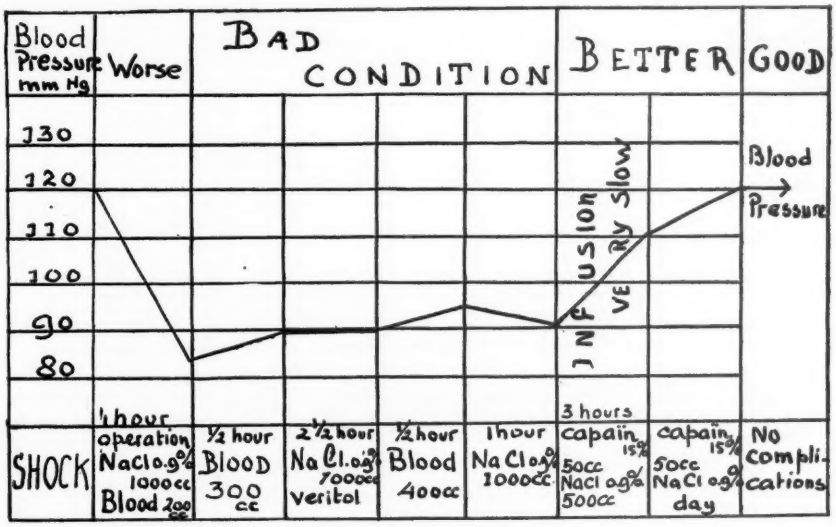

FIGURE II. Case XLI.

added. A heart tonic was given, too, as well as an injection of vitamin *K*. The patient recovered from her shock. The following days capain medication, together with the intravenous administration of some fluid, was continued; yet it became more and more certain that a fatal outcome was imminent, for the function of the liver showed a serious worsening. Urine production was sufficient, but the specific gravity showed no tendency to rise. The patient remained slightly apathetic. Suddenly she died on the fourth day after operation.

Summary.—In this case a large transfusion of blood did not improve the condition of shock; on the contrary, it gradually became worse after the transfusion, which certainly was not given too slowly. Capain effected a rise of blood pressure and at least the recovery from this shock condition. There was no difference between the speed of the various infusions. Thus special qualities of capain seem to be responsible for the favourable effects. Most probably these were due to its vasoconstrictor properties, in combination with its water-binding and its nutritive

sequently the patient was operated upon for wound cleaning under anæsthesia by nitrous oxide combined with ether.

After half an hour the patient's condition suddenly became worse and again blood was injected quickly. Pulse and blood pressure were unmeasurable. Immediately operation was terminated, and infusion was continued with 100 millilitres of 14% capain solution in one litre of 5% glucose solution. This amount was injected quickly; the blood pressure responded soon by rising. Both legs were treated by the traction method and splinted. There was continuous, though not severe, bleeding from the dressed wounds. Pressure bandages were applied. Within twenty minutes the capain glucose solution had been administered; the blood pressure was then 75 millimetres (systolic), yet the prognosis seemed to be bad. Another infusion of 100 millilitres of 14% capain solution in one litre of 5% glucose solution was given; the blood pressure rose to 90 millimetres of mercury (systolic) and 10 millimetres (diastolic). The pulse was good, the hæmoglobin value was 58%, and the erythrocytes numbered 2,770,000 per cubic millimetre. Medication was continued by infusing one litre of 5% glucose solution together with 100 millilitres of 14% capain solution.

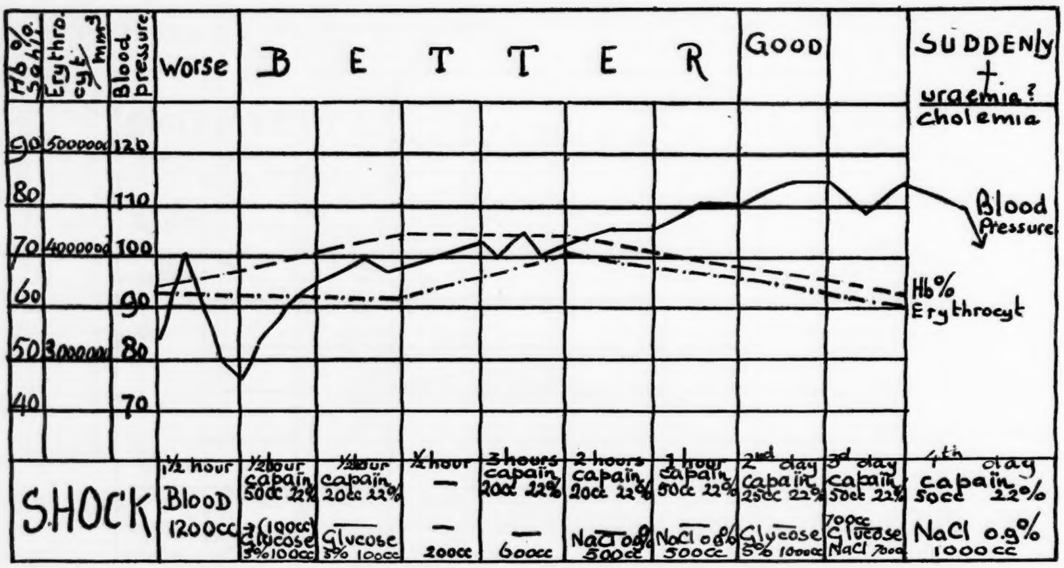

FIGURE III. Case IV.

qualities. The fact that despite this success the patient died is readily explained by the seriously damaged state of her liver as found at operation.

CASE LV.—A man, aged thirty-six years, was shot in both legs. Two hours later he was admitted to the clinic in a bad condition. There had been loss of blood from an extensive destruction of the right tibia with a lacerated soft-tissue wound, and an enormous destruction of the left crural bones with soft-tissue damage. There was continuous loss of blood. The blood pressure was 95 millimetres of mercury (systolic) and 60 millimetres (diastolic), the hæmoglobin value was 69%, and the erythrocytes numbered 4,200,000. The patient was in a state of shock. Capain saline infusion (100 millilitres of 14% capain solution in one litre of 0·9% saline solution) was given. Half an hour later (when 500 millilitres of the solution had been administered) the blood pressure was 110 millimetres of mercury (systolic) and 80 millimetres (diastolic), and the pulse rate was 70 per minute. Severe wound pain, unrelieved by morphine, was present. The blood pressure rose to 120 millimetres of mercury (systolic) and 80 millimetres (diastolic) within three-quarters of an hour. The condition remained the same. Injection of synephrin ("Sympatol") aggravated the pain. Then demerol ("Dolantin") was given intravenously (two millilitres) and the effect was clear: pain decreased, yet the blood pressure remained on the same level. The general condition was much better after this injection. After one and a quarter hours the infusion of capain saline solution was completed, and then blood was given. About 800 millilitres went in, in one hour. Sub-

These 2200 millilitres of fluid went in, in 80 minutes. The blood pressure then was 105 millimetres of mercury (systolic) and 40 millimetres (diastolic); the pulse was quick and well filled. However, continuous blood loss took place from the wounds; therefore "Sangostop" (a Dutch coagulation-promoting agent, consisting of pectins) was injected. Because of the anæmic condition 400 millilitres of blood were given in a quarter of an hour. Before this blood transfusion the hæmoglobin value was 35% and the erythrocytes numbered 2,060,000. In spite of this treatment, the condition worsened seriously and the blood pressure after transfusion measured only 60 millimetres of mercury (systolic) and 40 millimetres (diastolic). Again 100 millilitres of 14% capain solution in one litre of 10% glucose solution were given, but the patient died some hours later. Post-mortem examination revealed the already mentioned injuries, together with signs of death due to shock.

Summary.—In this case primary and secondary shock existed, combined with extensive soft-tissue destruction and consecutive serious loss of blood. Compensatory vasoconstrictor mechanisms had already subsided on the patient's admission to hospital. Synephrin worked invertedly by exaggerating irritability. Blood transfusion did not give satisfactory results. Capain fluid medication, however, showed more favourable effects on circulation, most probably due to the vasoconstrictor action of capain in combination with its other properties.

CASE LXI.—A woman, aged forty-seven years, was admitted to the clinic with a large scapular tumour that had

reappeared in spite of previous operations in 1932, 1941, 1942, and on July 26, 1943. This tumour proved to be malignant, as chondromata of the scapula often become after many recidivations. So an interthoracoscapular amputation of the arm was performed (after Berger) on December 15, 1944. Her blood pressure was 130 millimetres of mercury, systolic, and 80 millimetres, diastolic. Her hæmoglobin value was 80%. Operation was difficult because of many adhesions. A preventive infusion of saline was started before and continued during operation, which took about an hour. Blood loss was very moderate. The patient, however, got into a state of serious shock after the operation had been terminated; the pulse was filiform and quick. A blood transfusion was given, and 400 millilitres went in slowly. The blood pressure was not measurable, during the transfusion there was no improvement, and after one hour the condition was very bad. Then capain was given, 75 millilitres in 750 millilitres of 0·9% saline solution, which went in, in three-quarters of an hour, with good effect, for the blood pressure rose immediately after this medication, and after three-quarters of an hour the patient's condition was much better. Tension then measured 100 millimetres of mercury (systolic) and 50 millimetres (diastolic), the pulse rate was 132 per minute, and the respiration rate 32 per minute. After this a further 100 millilitres of 14% capain solution in one litre of 5% glucose solution were given. The blood pressure then rose to 110 millimetres of mercury (systolic) and 90 millimetres (diastolic). After the whole amount had been administered within ninety minutes the patient was in good condition. At one moment the blood pressure sank to 80 millimetres of mercury (systolic), but the peripheral vessels remained well filled. Capain medication was continued, and the patient's further recovery was uneventful.

Summary.—In this case, after severe surgical trauma, blood was given first, but probably too slowly. The subsequent capain-saline and capain-glucose infusions worked better, firstly by greater speed of infusion, and probably by vasoconstrictor action too. In this case there certainly was a combination of primary and secondary shock.

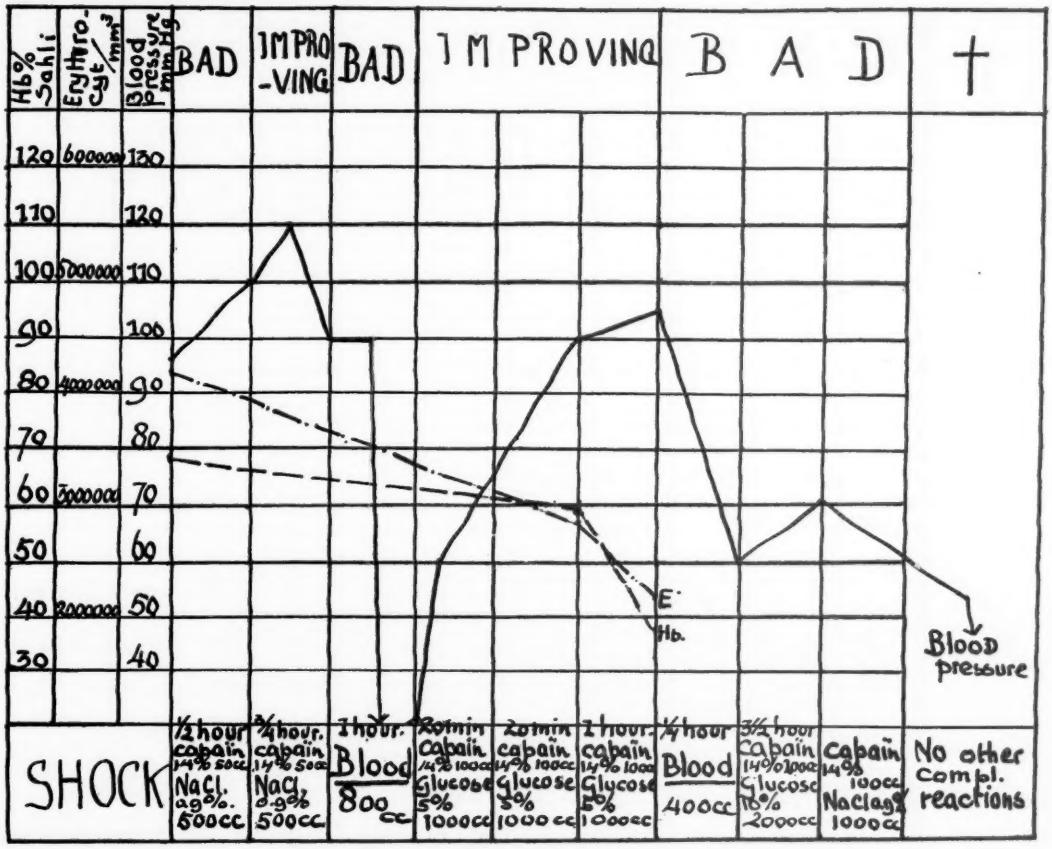

FIGURE IV. Case LV.

Discussion.

To summarize the results obtained in the treatment of some 70 shock patients with capain solution, the following statements can be made.

Generally speaking, the effect of intravenous infusion therapy with capain solution has been satisfactory. In those cases in which it failed, anatomical damage incompatible with life was usually the cause. In many of the successful cases blood had been infused previously with poor results.

With regard to possible toxic effects, the maximal dose should preliminarily be put at 75 grammes of capain, calculated as dry substance, as this is the largest amount which in our experience up to this moment did not arouse any suspicion. This quantity is closely related to the total amount of fluid that may be infused without ill effects, and that should not exceed about 6500 millilitres per patient. An overdosage of fluid seems to be more dangerous than an excess of capain. Of this fluid, not more than 2000 millilitres should consist of 5% glucose solution; infusion of more than this dose may contribute to serious complications, especially in cases of absolute or relative shortage in sodium chloride. It is of great importance to prevent such complications by giving capain-saline infusions first. The usual dosage is 100 millilitres of 15% capain solution to every litre of 0·9% saline solution. Preliminary reports make it advisable to start with at least about two litres of capain-saline solution. In future experiments, however, it should be borne in mind that

dissolving media such as the well-known Ringer or Ringer-Locke solutions may be preferable, provided they do not precipitate capain, thus giving rise to the danger of emboli. Although our own experiments in this respect have not yet given definite results, it is probable that the formation of dangerous precipitates can largely be prevented by adding small quantities of another innocuous (protective) colloidal substance to the capain solution.

We found the results of small blood transfusions disappointing. In these cases (about ten) the subsequent infusion of capain-saline solution brought the desired effect. Here the total amount of administered "water", staying long enough in circulation, seemed to be of the utmost importance. In cases of serious anæmia, however, the intravenous administration of blood is strictly indicated for restoration of the oxygen-carrying power.

In some cases the circulation recovered very rapidly. It was evident then that other than the purely hydromechanical mechanisms were responsible for the rise of blood pressure. These clinical observations made us suspect that capain possessed marked vasoconstrictor activities: this was confirmed by experiments on animals. This vasoconstrictor activity probably contributes a great deal towards the successful action of capain in shock treatment. For, on observing our patients, we became more and more convinced that in many cases of traumatic shock a neurogenic primary shock component with reflex vasodilatation forms an important part of the whole complex of ætiological shock factors. In our opinion vasodilatations of neurogenic origin even play a decisive role in the pathogenesis of traumatic shock. They may occur in more or less extensive areas. Sometimes a condition of primary shock with bradycardia will initiate the whole complex of shock components that will result in severe secondary shock. The prognosis of such a combination is serious, because the primary shock, with its accompanying low blood pressure, will undoubtedly accelerate the onset of secondary shock symptoms. In most cases, however, such an initial phase of primary shock is camouflaged by compensatory vasomotor reactions. Moreover, it seems quite possible that in one part of the body reflex vasodilatation is maintained by stimuli coming from the wound areas, whereas in other parts of the body a compensatory vasoconstriction succeeds in maintaining blood pressure at the normal level. In a given case it is by

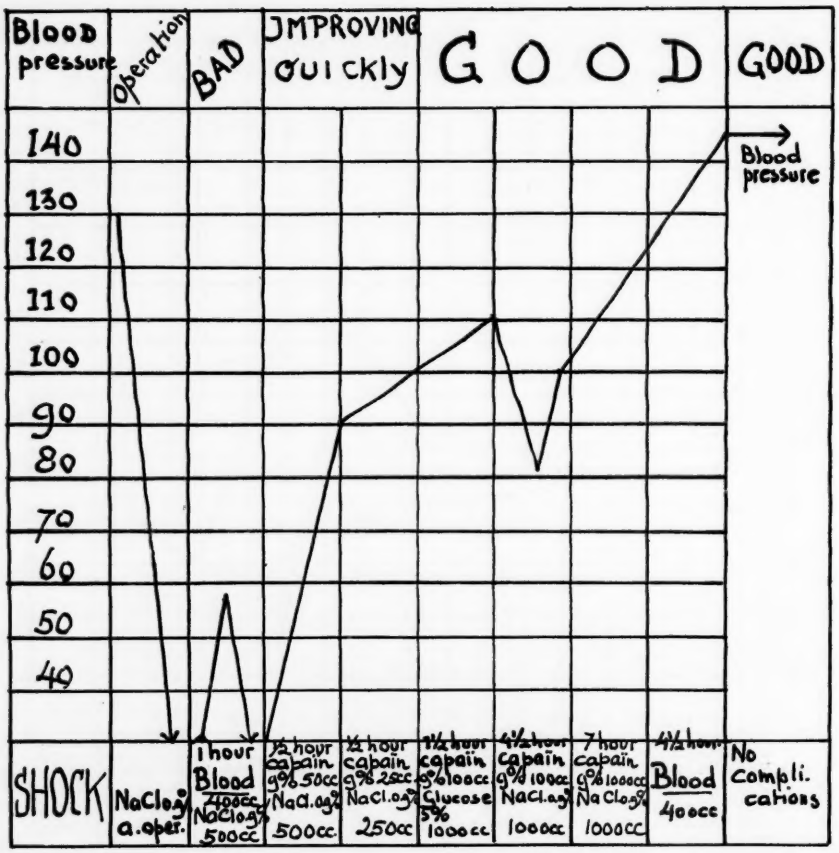

FIGURE V. Case LXI.

no means certain that the initial primary shock components will subside spontaneously, so it will be wise to combat them as soon as diagnosis can be made. Our clinical experiments proved that rapid intravenous injection of concentrated capain (50 millilitres of 15% solution) may have favourable effects in such cases by its vasoconstrictor action, which probably acts on peripheral vessels too. In cases in which the diagnosis of primary shock cannot be made, intravenous infusion of a more dilute solution of capain in physiological saline in sufficient quantities is the safest way. When vasodilatation exists anywhere in the body, the vasoconstrictor activity of capain may combat this.

Secondary shock, of course, should be treated by infusion of larger volumes of diluted capain solutions. In some cases in which these had been administered, post-mortem examination yet revealed absence of excessive œdema in the wound areas. More experiments and clinical observations will be required to decide whether really permeability-

decreasing (antihistaminic?) activities or mechanical mending effects on leaking capillary walls, damaged by shock, do exist. It is generally accepted that histamine intoxication is one in the above-mentioned complex of ætiological factors in secondary shock, and probably the most serious one. It is known, too, that some amino acids exert antihistaminic properties. Perhaps the introduction of the modern antihistaminics may further add to a more efficient therapy in similar cases.

Capain may, owing to its vasoconstrictor action, support an insufficiently functioning adrenergic compensation mechanism, which is responsible for the vasoconstrictor reactions in order to maintain blood pressure at a normal level when much fluid has disappeared from circulation. To combat regional vasodilating stimuli coming from the wound areas we can use Wishnevsky's method of blocking the cervical sympathetic and vagus nerves by local anæsthetics, which notably lowered mortality from shock during the last war in Russia.

On the battlefield it will be possible to combat primary shock components by rapid intravenous injections of concentrated capain (15% to 20% solution), eventually repeated. Moreover, these injections of powerfully water-attracting material will help to prevent the development of secondary shock by preventing or delaying the loss of fluid out of the circulation. Patients will arrive in a better condition at the field hospitals, where slow-infusion therapy with larger volumes of fluids is possible. Apart from the efficient shock-combating power of capain solutions, as already mentioned, the use of concentrated capain solution in the front line is likely to have other advantages.

Chilly reactions were sometimes observed, but never were they followed by more serious complications. The chills were abolished at once by intravenous injection of one or two millilitres of demerol ("Dolantin"). They are not of pyrogenic origin; on the contrary, in many cases their onset, without hyperthermia, marked the recovery of circulation.

Awakening effects comparable with those produced by nikethamide were sometimes observed, and a few times we saw reactions resembling those to digitalis or to nitrites.

Some hypoproteinæmic patients responded with a quick recovery after treatment with capain, so it seems probable that rapid synthesis of blood proteins from this material may occur.

Vomiting was seen only rarely; when it occurred it was not quite clear whether it was really due to capain and its constituents such as glutamic acid.

It seems preferable not to apply capain treatment in shock when the kidneys are known to be diseased, because of the known deleterious influence of some amino acids, such as serine, on damaged renal tissue.

Summary.

Summarizing, we can say that in our experience the treatment of traumatic shock with capain was satisfactory in nearly every instance. It can be easily manufactured in every amount wanted, and the technique of administration is very simple. Capain proved its value in cases of shock with serious initial primary shock components, as well as in cases in which vasodilation of other origin endangered life. It maintains its water-attracting and water-binding power long enough intravascularly to combat secondary shock with success; intravenous injections of concentrated capain solutions may have life-saving effects on the battlefield by combating primary vasodilatation and by delaying the secondary loss of fluid out of circulation.

Capain helps economy in the use of blood and of plasma, which is now restricted to special indications.

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Reviews.

INJURIES TO THE HEAD.

It is not surprising that a work of the excellence and usefulness of Rowbotham's "Acute Injuries of the Head" should now have entered a third edition.[1]

In the five years that have elapsed since the second edition appeared no great increase has accrued to our knowledge of head injuries or their treatment. The late war made its great contribution in a further understanding of pathology and in its revolutionary control of infection. Although knowledge has not increased greatly, the general scope and presentation of this book have improved to a considerable degree. It is without doubt the most complete and best work of its kind, and as such should be readily available to any one called upon to treat head injuries, that is, every doctor in general or surgical practice. Even the specialist neurosurgeon could profitably turn over its pages and gain stimulus and help.

The pathological section is a fine and useful contribution, particularly in a community like our own where the workings of the *Coroner's Act* prevent the post-mortem demonstration of head injury effects to students who thus never have a chance to see the pathological condition for themselves, and likewise deprive the responsible surgeon from obtaining a ready check on his clinical judgements. This is particularly tragic, as there seems to be no subject so shrouded in mystery and so beset with difficulties to the average doctor.

It is in such case that he may turn to Rowbotham's book and gain light on his problem. There is here much wise advice on early diagnosis and treatment, but more important even, on the early and effective rehabilitation of the injured.

One would think that Mr. Rowbotham had enjoyed writing this book, for he has infused into it a living and almost tense interest which makes for easy reading.

INTERNATIONAL STATISTICAL CLASSIFICATION OF DISEASES.

Volume I of the "Manual of the International Statistical Classification of Diseases, Injuries and Causes of Death" has been compiled under the auspices of the World Health Organization by a group of experts on health statistics.[2] The volume is really the sixth revision of the International List of causes of death. The purpose of the manual is to secure such uniformity in morbidity and mortality statistics as can be achieved throughout the world.

A decimal system of numbering has now been adopted in which the detailed categories of classification are designated by three digit numbers. This numbering system is a departure from the combined number and letter subdivisions used in the previous International Lists.

Not every disease condition receives a particular number, but there is a category in which every condition can be placed; this has been achieved by selective grouping. Disease groups have been given numbers from 001 to 799, with suggested subdivisions in many instances denoted by a fourth digit, for example, 550, acute appendicitis; 550·0, acute appendicitis without mention of peritonitis, perforation

[1] "Acute Injuries of the Head: Their Diagnosis, Treatment Complications and Sequels", by G. F. Rowbotham, B.Sc. (Manchester), F.R.C.S. (England), with a foreword by Professor Norman M. Dott, C.B.E., M.D., Ch.B. (Edinburgh), F.R.C.S. (Edinburgh); Third Edition; 1949. Edinburgh: E. and S. Livingstone, Limited. 9¾″ × 6¼″, pp. 504, with 259 illustrations. Price: 35s.

[2] "Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death: Sixth Revision of the International Lists of Diseases and Causes of Death, Adopted 1948", Volume I; 1948. Geneva: World Health Organization. 9½″ × 6¼″, pp. 377.

or rupture; 550·1, acute appendicitis with perforation or rupture.

Accidents, poisoning and violence are given a dual classification with numbers ranging from 800 to 999, with the letter E (E800-E999) denoting external cause, and the letter N (N800-N999) indicating the nature of the injury. The manual contains guiding rules to assist the compiler of morbidity and mortality statistics in the use of the international classification.

Statistics of this nature provide the necessary numerical data by which the health conditions and needs of the various peoples of the earth may be evaluated. So the World Health Organization offers this book, that it may be not merely an object of interest to medical statisticians, but that it may be the means for collecting into a common pool of knowledge, information which may be, in time, of benefit to all mankind.

A YEAR BOOK OF PÆDIATRICS.

THE editor of "The 1949 Year Book of Pediatrics", Henry G. Poncher, has developed the making of editorial comment considerably beyond that in previous corresponding volumes and indeed that in others of the Year Book series.[1] Many sections are preceded by a concise review of significant trends during the past year, mention is sometimes made of trends within the editor's knowledge which have not yet appeared in the literature, an attempt is made to assess the ultimate importance of some of the newer developments, and additional references may be recommended to help the reader in making his own assessment. On the other hand no comments are made just for the sake of saying something; many abstracts appear without comment, and the editorial notes are always to the point. An introductory article, "The Year in Pediatrics", is informative and brief. The abstracts presented are grouped under the same headings as those of the previous volume and have been selected from journals received during the period July, 1948, to July, 1949. Australian work noted includes Grantley Stable and Iris G. Philpott's report on an epidemic of gastroenteritis in infants, Murray Clarke's congress paper on intestinal obstruction in infants, the investigations of Rachel Jakobowicz, Vera I. Krieger and R. T. Simmons into the value of the Coombs test in detection of isosensitization of the newborn, and of E. M. A. Day into the urinary excretion of 17-ketosteroids and of corticosteroid-like hormones by the newborn infant, and Kate Campbell's discussion of intracranial disorders of the newborn associated with birth.

EXPERIMENTAL SURGERY.

NOT the least important point about Markowitz's book is that it is concerned only with "experimental surgery" in animals.[2] It includes a comprehensive, interesting and well-illustrated account of surgical craftsmanship, including knot tying, suturing and the use and handling of various instruments.

As the author writes: "It is not the best course in surgery to insist that a student ordinarily have a proper training in everything but surgery, and is allowed only to hold their retractors for the final year of his course, and perhaps open or close an abdominal incision or two." Further, to demand from a would-be surgeon a post-graduate knowledge of the fundamental sciences at the expense of any knowledge of surgical craftsmanship is obviously wrong.

This book is a text-book for the subject of experimental surgery, whereby in many United States hospitals the students and nurses gain, on experimental animals, their first experience in pre-operative and post-operative treatment, theatre room technique, surgical craftsmanship and anæsthesia. It contains full details of all aspects of surgery in experimental animals, and should be indispensable in physiological laboratories, and of great help to medical students and recent graduates who seek to educate their hands in basic surgical procedures—and not at the expense of the patient.

[1] "The 1949 Year Book of Pediatrics (July, 1948-July, 1949)", edited by Henry G. Poncher, M.D., with the collaboration of Julius B. Richmond, M.D., and Isaac A. Abt, M.D.; 1949. Chicago: The Year Book Publishers, Incorporated. 7" x 4½", pp. 562, with 120 illustrations. Price: $4.50.

[2] "Experimental Surgery: Including Surgical Physiology", by J. Markowitz, M.B.E.; Second Edition; 1949. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson. 9" x 6", pp. 564, with 330 illustrations. Price: 52s. 6d.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Tuberculosis: A Global Study in Social Pathology", by John B. McDougall, C.B.E., M.D., F.R.C.P. (Edinburgh), F.R.F.P.S. (Glasgow), F.R.S.E.; 1949. Edinburgh: E. and S. Livingstone, Limited. 9¾" x 7", pp. 468, with few illustrations. Price: 32s. 6d.

The book has two aims—to present information on infection, morbidity and mortality from tuberculosis, and to analyse factors considered to have a bearing on the extent and severity of the problem.

"Selective Partial Ablation of the Frontal Cortex: A Correlative Study of its Effects on Human Psychotic Subjects", by The Columbia-Greystone Associates; edited by Fred A. Mettler, M.D., Ph.D.; 1949. New York: Paul B. Hoeber, Incorporated. 10" x 7", pp. 540, with few illustrations. Price: $10.00.

A cooperative report of a cooperative endeavour—describes a study of 48 patients.

"Lord Lister: His Life and Doctrine", by Douglas Guthrie; 1949. Edinburgh: E. and S. Livingstone, Limited. 8" x 6½", pp. 142, with illustrations. Price: 15s.

An attempt to view the achievement of Lister in the light of modern knowledge.

"The Nature and Technique of Understanding: Some Fundamentals of Semantics", by Hugh Woodworth; 1949. Vancouver: The Wrigley Printing Company, Limited. 8½" x 5½", pp. 156. Price: $4.00.

An attempt to discover what is wrong with our thought, that it should promise so much and produce so little.

"The Conduct of Life Assurance Examinations", by E. M. Brockbank, M.B.E., M.D., Vict., F.R.C.P.; Third Edition; 1949. London: H. K. Lewis and Company, Limited. 8½" x 5½", pp. 180. Price: 12s. 6d.

One of the "General Practice Series"—deals with the examination and with impaired lives.

"A Short History of Physiology", by Kenneth J. Franklin, D.M., F.R.C.P.; Second Edition; 1949. London: Staples Press, Limited. New York: Staples Press, Incorporated. 8¾" x 5½", pp. 154, with a few illustrations. Price: 10s. 6d.

Deals with the development of human physiology from the sixth century B.C. to the end of the nineteenth century A.D.

"Infant Nutrition: Its Physiological Basis", by F. W. Clements, M.D., D.P.H., D.T.M.; 1949. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall, Limited. 8¾" x 5½", pp. 260. Price: 21s.

An introduction to the subject based on relevant and related facts culled from various sources with personal observations.

"Personality Maladjustments and Mental Hygiene: A Textbook for Students of Mental Hygiene Psychology, Education, Sociology and Counselling", by J. E. Wallace Wallin, Ph.D.; Second Edition; 1949. New York, Toronto and London: McGraw-Hill Book Company, Incorporated. 9" x 6", pp. 598. Price: $5.00.

The aim of the book is to introduce the student to the problems of mental health and mental hygiene.

"A Guide to the Diagnosis of Occupational Diseases: A Reference Manual for Physicians", compiled jointly by the staffs of the Industrial Health Division Department of National Health and Welfare and the Division of Industrial Hygiene Department of Health for Ontario; 1949. Ottawa: The King's Printer and Controller of Stationery. 9" x 6¼", pp. 322. Price: $1.00.

This book is intended to present only a brief summary of current knowledge of the diagnostic features relevant to occupational diseases.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE NATURE AND MEANING OF PAIN.

WHEN, in Shakespeare's "Macbeth", word comes to Macduff of the murder of his wife and children, Malcolm exhorts him to "dispute it like a man". Macduff replies: "I shall do so", and then adds, speaking for us all: "But I must also feel it as a man." Whatever our other reactions to pain in any of its forms, the common heritage of the sons of men is to "feel it as a man". What then is it that we "feel"?—and here we address ourselves only to physical pain—what is pain, its nature and its meaning?

Those who have studied physical pain are the most reluctant to attempt a definition. Sir Thomas Lewis has stated that the attempt could serve no useful purpose. Others have offered limited definitions appropriate to a particular approach. H. G. Wolff and J. D. Hardy[1] point out that until the end of the nineteenth century pain was considered to be exclusively a feeling state. Later, with the discovery of "special anatomic equipment and mechanisms", attention was focused on the perceptual aspects of pain. It then became clear, they continue, that pain is a specific sensation, and yet, because of its intimate linkage with strong feelings and other reaction patterns, the latter may be dominant in the experience. Reactions to pain may be modified by conditioning experiences and strong beliefs. The common analgesics, in addition to raising the pain threshold, also have a major function in changing reactions, attitudes and feelings.

> Thus, recent evidence supports the old view that the "quale" or feeling state is, to the one who suffers, perhaps the most relevant aspect of pain. Yet it supports as well the conclusion that pain is a specific sensation with its own structural and functional properties. It becomes apparent that these two concepts do not oppose each other: both represent attempts to formulate distinct but fundamental aspects of the pain experience.

A further analysis of pain is attempted by Harold Balme in his book "The Relief of Pain". He distinguishes three separate elements in every pain-sensation: first, an acute sense of physical distress, a raw feeling of "hurt"—primitive and specific, telling of something interfering with physical well-being; secondly, a sensory impression, of a critical and discriminating character, which enables us to locate both the site and the nature of the lesion; thirdly, an emotional upheaval, varying with our reaction to the cause of the injury. The three elements occur in varying proportion and relative importance, but their consideration illuminates the comment of Leriche (quoted by Balme) that physical pain is "the resultant of the conflict between a stimulus and the whole individual". The thought is taken a step further with Sir Charles Sherrington's description of physical pain ("Man on His Nature", page 286) as "the psychical adjunct of a protective reflex". Admittedly most protective reflex actions, as pointed out by G. W. Theobald,[1] are set in motion by non-painful stimuli, and the intensity of pain bears little or no relation to the amount of tissue damage. But for the moment it is sufficient to note that in response to a harmful stimulus the individual reacts and experiences pain. Investigation of the structural basis of the reception, transmission and appreciation of the stimulus still goes on. Writing ten years ago, Balme stated that the majority of physiologists accepted the view that there were "additional nerve-fibres solely concerned with the transmission of painful impulses" and that "painful impulses reach the sensorium by a different route from that followed by ordinary tactile sensation". More recently Professor E. D. Adrian[2] was reported as saying that ambitious attempts to distinguish sharply between the sensory apparatus of pain on the one hand and that for finely graded discriminatory sensations on the other had had to be given up, and the tendency was to think of sensory fibres and endings as forming more or less a continuous series in which sharp distinctions could not be enforced; pain mechanisms employed few if any of the largest nerve fibres, which seemed to be concerned with touch and muscle sense, but there was no one size or variety of fibre devoted to pain. So that particular question stands, though many will feel that the position is negative and not wholly satisfactory. Of the central mechanism of pain, much that is suggestive has come to light, especially with advances in brain surgery. The relative rôles of the thalamus and the cerebral cortex are being sorted out. Thus W. Freeman and J. W. Watts,[3] well-known workers in this field, are able to write:

> Prefrontal lobotomy has a beneficial action upon pain whether it is primarily mental or primarily physical. It does not interfere with the perception of pain, but rather with the evaluation of pain. It does not relieve the pain, but rather the disabling reaction to pain, the fear of pain. It does so apparently by eliminating the emotional component arising from the thalamus.

These investigators and many others are gradually uncovering the mechanism of pain and we can scarcely question the existence of that mechanism though opinions differ as to its specificity. The question of its meaning then arises; though we avoid the controversial term "purpose", we may still ask if it has a function, if it does anything of biological value to the individual. Lord Moran,[4] in a thoughtful discussion of the question, emphasizes the importance of differentiating between deep and superficial pain. Pain arising in superficial structures is accurately localized and has certainly a protective value; this cannot be said about pain of deep origin, which appears to serve no useful biological function—any value

[1] *Physiological Reviews*, April, 1947.

[1] *The Lancet*, July 16, 1949.
[2] *British Medical Journal*, September 20, 1947.
[3] *Annals of Internal Medicine*, April, 1948.
[4] *The Practitioner*, February, 1948.

it may have in modern clinical diagnosis scarcely comes under this heading. Where, however, the pain and its source can be related, the individual stands to benefit, though some damage may already have been done and the pain may continue beyond a useful period; the benefit will range from reflex stimulation of withdrawal from the source of harm (an activity in which pain, if experienced, may appear largely redundant) to the storing of warning memories and the development of elaborate plans to prevent or mitigate subsequent similar experiences. Pain has thus, as Adrian has said, a far greater survival value for man than it could have for creatures with smaller brains. The same will apply, of course, to what Sherrington refers to as its "positive counterpart, training by reward, a seeking to repeat remembered satisfaction". Mind then appears to be the essential concomitant of pain. Protective reflexes, Sherrington states, afford a measure of protection.

> Where they are yoked with mind . . . their sensual accompaniment is rich in affect, an acute psychical urge, in short "pain". This urge reinforces and amplifies the measure of protection and relief the pure reflex act affords.

Thus we come back to his description of physical pain as "the psychical adjunct of a protective reflex". Adrian has pointed out that other sensory functions can play a part in protection. The central nervous system of man is so highly organized as to make him uneasy in certain sensory situations—noise, for example—and to tend to make him avoid them, without any actual experience of pain. Pain, however, involves the mind directly in the threatening situation which is the primary concern of the body. The mind then not only cooperates in the body's protective efforts but urges it on; in Sherrington's words:

> In short, under the rubric "pain" we meet mind moving matter to help mind in mind's distress. Mind invoking the body to do something and, in spite of the eternal psycho-physical difficulty, effectively. "Pain" seems to pay no heed to that old dilemma. My raging tooth drives me to the dentist as if it operated my motion thither.

Current Comment.

POLIOMYELITIS IN THE ARCTIC.

OUR urgent need to understand the behaviour of poliomyelitis makes all competent epidemiological studies of the disease worthy of attention. Peculiar interest, however, attaches to a report of a widespread epidemic of acute poliomyelitis among Eskimos in the Eastern Arctic of Canada during the autumn of 1948 and the winter of 1949; it is presented by J. D. Adamson, J. B. Moody, A. F. W. Peart, R. A. Smillie, J. C. Wilt and W. J. Wood.[1] The epidemic was one of the most northerly on record and among the very few known to have occurred among Eskimos. The area involved is on the western coast of Hudson Bay almost 1000 miles north of Winnipeg. The epidemic occurred during the coldest part of an unusually cold winter in one of the coldest regions of the Arctic. Its effect was devastating; in one locality (there were two separate localized outbreaks in the epidemic) over 5% of the total Eskimo population (275) died from poliomyelitis, 14% were paralysed and probably at least 50% had symptoms of the disease. In the other locality the effect was nearer to that expected in a white community: 0·8% of the population (500) died, and 4·2% were paralysed; no estimate can be made of the number with symptoms short of paralysis. The spread of the disease appears to have been largely through the medium of clinically healthy carriers; the probable links in the chain of infection appear to be complete in all cases, being particularly clear for certain isolated infections. Individuals of all ages were affected, except those under three years; only two children aged three and four years were involved. The brunt of the disease, which monkey inoculation showed to be due to the poliomyelitis virus, fell on the lower part of the spinal cord; death was caused by gradually ascending paralysis. It is notable that the main effect of this ascent was the paralysis of respiratory muscles with a short period only of respiratory distress and cyanosis before death; very few signs appeared of meningeal, bulbar or cerebral involvement. It is suggested by Adamson *et alii* that the tendency of the virus to select the lower part of the spinal cord is an indication of gastro-intestinal rather than respiratory tract infection. Other evidence is lacking to differentiate between these two possible routes of infection; the personal habits and hygiene of the Eskimo provide excellent oportunities for both. The climatic conditions rule out any possible insect vector except the louse. Perhaps the most remarkable feature of the epidemic was the explosive outbreak at Chesterfield with the high death and paralysis rates already mentioned. It lasted only twenty-one days and all 14 deaths occurred in a period of eleven days; in 10 cases the period from onset of paralytic symptoms to death was only four, five or six days. The spread through the community was quite unlike that normally seen in white communities, conforming rather to that of an acute and highly infectious disease. Three circumstances are suggested to account for the violence of this outbreak in contrast to that in the other locality with its death rate of less than 1%: firstly, the more northern locality and lesser contact of the natives with white people, which may explain a lower resistance; secondly, crowding into camps and igloos because of the winter season with less ventilation and long hours of intimate contact; thirdly, the great tendency of natives to congregate about the trading post, the mission and the hospital. Such a combination of circumstances favourable to epidemic spread will, fortunately, not always be found in a susceptible population, but Adamson and his colleagues feel that comparable damage may well be done in other Eskimo communities, and thorough quarantine restrictions have been imposed. These contrast with the rather half-hearted attempts at control usual in white communities and serve to stress the seriousness with which we should regard the possible spread of poliomyelitis to any relatively isolated native community.

[1] *The Canadian Medical Association Journal*, October, 1949.

REFLEX TROPHIC CHANGES FOLLOWING CORONARY OCCLUSION.

IT is fifty years since Sudeck described the peculiar form of osteoporosis which may follow trauma. There had previously been some literature on this subject, and there has been a considerable volume of writing since. A. W. Hilker in an article on what he calls "the shoulder-hand syndrome", extends the ambit of these trophic disturbances to cardiac accidents, in which the trauma is intrinsic, but, of course, none the less damaging.[1] He points out that de Takats has brought causalgia into the same post-traumatic category, and that recently distrophies of a type usually connected with trauma have been described as due to other causes. In the present instance we are concerned with dystrophy of the upper extremity, and some authors have found it to occur after hemiplegia, *herpes zoster* and myocardial infarction. In all a great variety of causes have been recognized, and to explain the mechanism the concept of an internuncial chain of neurons has been invoked. Such a chain is described as a network of interconnecting neurons extending over a number of spinal segments of the cord, linking incoming impulses with the motor neurons of the anterior horn cells and the sympathetic system. Hilker has collected eleven instances of the shoulder-hand

[1] *Annals of Internal Medicine*, August, 1949.

syndrome after coronary occlusion. Though several series of this complication or sequel collected by others suggest that it may occur in from 10% to 22% of all patients suffering myocardial infarction, he thinks that it is probable that early and reversible ischæmic changes may be seen in one or both upper limbs in a considerable though unspecified proportion. His patients showed evidence of affection of either or both of the upper extremities within three to twelve weeks after a severe coronary occlusion. There was a definite correlation between the intensity and duration of the cardiac attack and the occurrence of the trophic disturbance. The early symptoms were either pain in the shoulder or tenderness and swelling of the hand. When the hand was affected it was usually red or bluish, and as the condition progressed it became cold, the skin became thin, and the muscles atrophied. Radiographic examination showed trophic changes in the bones of the metacarpus and hand. The shoulder when affected was painful on movement, which soon became limited, and here, too, the upper end of the humerus showed atrophic changes. These lesions were not necessarily irreversible, but when this stage was reached a certain degree of permanent disability was very common. Sometimes the lesion was further progressive, and serious crippling took place. An affected shoulder became fixed and remained a source of discomfort and disability, and an affected hand came to resemble the condition familiar in severe forms of Sudeck's atrophy, with stiffness, atrophy and lastly immobility. Hilker's most severely affected patients who unfortunately reached these end states had serious infarctions of the anterior part of the heart, and also had evidences of congestive failure. He attempts to analyse the reasons for this crippling sequel of a potentially serious accident, and points out that although disuse may account for some part of the condition, the end result may be far too severe for that to be the only cause, and, further, it is hard to explain the latent period, which is often a number of weeks or more. The observed relation between the degree of cardiac affection, and indeed of vascular affection, cannot be ignored, nor can the finding that residual myocardial insufficiency seems to aggravate and perpetuate the syndrome. It is easy to understand that disturbed efficiency of the circulation will lower metabolism in general, but unless some neural physiological lesion is invoked, it is hard to picture the mechanism. However, it is not necessary here to pursue the argument further, as it is sufficient to draw attention to an important sequel of myocardial infarction, which has, of course, been widely recognized before, but has not received the attention it merits.

GASTRIC ACIDITY AND GASTRIC ULCER.

THERE is strong evidence of a causal connexion between peptic ulcer and the action of gastric juice containing abnormal amounts of hydrochloric acid. Part of this evidence is based on pathological data relating proved ulcers with sites exposed to acid secretion. Part depends on animal experiment, but although this is very convincing there are some links missing in the chain of evidence in the human subject. A. H. James and G. W. Pickering, introducing an account of some experimental work on man, remark that this evidence depends on test meals and examination of stomach contents withdrawn during the night.[1] The picture given by these means, as they point out, is fragmentary. After some experience with nocturnal aspiration they discontinued it, because they were uncertain if differences found were due to real differences in secretory activity or to variations in the efficiency of methods for recovering fluids entering the stomach. Therefore they began an inquiry into the pattern of gastric activity throughout a period of twenty-four hours. A Ryle's tube was placed and kept in a verified position, and small samples were removed every half hour during the day, and every hour during the night. Control subjects not suffering from peptic ulcer were included in the tests. Twenty patients with gastric ulcer and twenty-three with duodenal ulcer were investigated. Ulcer craters were demonstrated radiologically. Care was taken to exclude gastric carcinoma from the former series; this was done by surgical confirmation, gastroscopic examination, and clinical and radiological observation over a considerable period. The control series consisted of people with totally different maladies; none of them were seriously ill. The degree of acidity was expressed in terms of the pH, determined by the electrometric method; a close parallel between the pH and hydrogen ion concentration of hydrochloric acid in aqueous solutions at 25° C. was established by experiment. Sources of error by sampling were eliminated as far as possible by comparing samples aspirated from apertures in the tube some distance apart, and also from tubes placed in different sites in the stomach, as verified by X rays. By these means the authors were able to collect a more representative set of data about intragastric acidity in peptic ulcer than has hitherto been obtained. They have analysed their results in detail, and have been careful in drawing conclusions. The findings may be summarized as follows: subjects with duodenal ulcer yielded more gastric contents during the night than normal subjects, on the whole, and those with gastric ulcer yielded less. In duodenal ulcer the mean minimum acidity in twenty-four hours was greater than the normal, and the duration of high acidities was increased; in general, too, the level of acidity was higher, was less efficiently neutralized by food, and was maintained at night after food had left the stomach. In gastric ulcer the findings were different in some respects. The duration of low acidities was increased and that of high acidities was normal. In 16 out of 23 cases the curve of gastric acidity fell to, or near, neutrality at night when food had left the stomach. Evidence was obtained that the neutralization of gastric contents at night in patients with gastric ulcer was due to cessation of acid secretion. Many observers have found, incidentally, that a gastric ulcer will often heal with a patient in bed, and on a suitable diet, but without administration of alkali. The authors conclude that whereas their findings agree with the hypothesis that duodenal ulcer is caused by the action of abnormal degrees of acidity acting over abnormally long periods of time, a similar agreement cannot be demonstrated with gastric ulcer. They suggest that gastric and duodenal ulcer have a different pathogenesis.

It is interesting to compare this work with some more of the observations of Stewart Wolf, in which he summarizes evidence relating life situations and emotional response to peptic ulcer.[1] He describes several case histories in great detail, and relates the clinical events with the passing events of the patients' lives, and gives records of acid secretion, motor activity of the stomach, gastroscopic appearances and psychological reactions. In these studies more attention has been paid to the physical and chemical changes occurring in the secreting mucosa of the stomach during the abreaction deliberately induced in the patients than to evidence of the exact anatomical site of the ulcer. It is perhaps significant that a history of an acute episode such as gastro-intestinal bleeding was given as a rule.

Here, then, we have sharply contrasted experiments—one set calmly carried out in a hospital, or at least as calmly as the presence of a Ryle's tube will allow; the other set reproducing the crises of life which have apparently caused a severe fronto-thalamic reaction, whose stress fell on a vulnerable stomach or duodenum. Wolf points out that personal conflict may express itself by gastric hyperfunction, apparently mediated by vagus innervation, and the local result may then be a lowering of the pain threshold in the stomach, and an increased fragility of the mucous membrane.

If we may indulge in a little speculation, it would seem possible that broad study of the psychological background, including the personality, together with the peripheral neural and chemical mechanism, may lead down to a point where local anatomical and physiological differences yield different pathological results in the stomach and duodenum, which yet have a common source.

[1] *Clinical Science*, Volume VIII, Number 3, 1949.

[1] *Annals of Internal Medicine*, October, 1949.

Abstracts from Medical Literature.

RADIOLOGY.

Radiological Features of Neurofibromatosis.

JOHN F. HOLT AND EDWIN M. WRIGHT (*Radiology*, November, 1948) state that the incidence of bone involvement in neurofibromatosis is considerably greater than the figure of 7% commonly quoted in the literature. Furthermore, because of the wide variety of bone changes which may occur, they believe that neurofibromatosis should be considered more frequently in the differential diagnosis of obscure skeletal abnormalities. Likewise radiological skeletal surveys of patients with known von Recklinghausen's disease are apt to show entirely unsuspected osseous defects. In general the bone lesions in neurofibromatosis may be classified as erosive defects, scoliosis, disorders of growth, bowing and pseudarthrosis of the lower part of the leg, intraosseous "cystic" lesions, and associated congenital anomalies. In this paper, each type of osseous lesion is considered separately, and various facts and theories are reviewed in regard to the mechanism by which each is produced.

Polyostotic Fibrous Dysplasia and Dyschondroplasia.

L. R. SANTE, W. BAUER AND R. M. O'BRIEN (*Radiology*, November, 1948) correlate the radiological and pathological findings in polyostotic fibrous dysplasia and dyschondroplasia and discuss certain radiological variations between these two diseases and other conditions of fibrocystic appearance with which they may sometimes be confused. The authors state that polyostotic fibrous dysplasia is easily differentiated from other types of bone involvement if the extraskeletal lesions, abnormal skin pigmentation and sexual precocity in the female are present. When fully established the radiological findings are in some respects similar to those of generalized *osteitis fibrosa cystica* (von Recklinghausen's disease). In the developing stage there is one outstanding difference: in generalized *osteitis fibrosa cystica* there is extreme osteoporosis due to the hyperparathyreoidism which is invariably present; in polyostotic fibrous dysplasia this is lacking. Osteoporosis is responsible for the extreme degree of deformity from bending of the bones which sometimes occurs in generalized *osteitis fibrosa cystica*; in polyostotic fibrous dysplasia deformity, if any, is local, being due to enlargement of individual lesions; the bones become weak from thinning of the cortex and may fracture, but bending as a result of softening is not encountered. In polyostotic fibrous dysplasia the expanded areas in the cortical and subcortical regions are not true cysts, but are filled with fibrous tissue; they are sharply delineated by the bony structure. The intervening bone appears normal, and there is no osteoporosis or other evidence of parathyreoid involvement. In generalized *osteitis fibrosa cystica*, the vacuolated areas are true cysts in the bone, arising also in the cortical structures, having at times definite lining membranes of fine fibrous tissue and containing clear straw-coloured or blood-tinged fluid. The remainder of the bony structure shows osteoporosis, and between the trabeculæ there is extensive fibrous tissue. Differentiation must be made from other rarer types of similar-appearing bone lesions, such as Ollier's dyschondroplasia. In this condition X-ray examination discloses irregularly distributed rarefied areas in the diaphyses with unilateral expansion of the metaphyses and thinning of the cortex. Either a single bone may be involved or there may be manifestations of involvement in a number of bones. The origin of Ollier's disease in the cancellous structure in the region of the metaphysis and the development of the lesions of fibrous dysplasia in the cortical regions of the shafts of the long bones should aid materially in differential diagnosis. Moreover, in Ollier's disease the radiogram reveals longitudinal bony strands of preserved bone trabeculæ radiating fan-like at the ends of the involved bones, with the rarefied areas caused by cartilage deposits in between. Small, heavily calcified globular bodies are observed within these mottled areas. Both of these findings are very important in differential diagnosis. The condition arises in early childhood during the period of bone growth. As the bone develops, the inhibition of bone growth in the involved portions of the diaphyses and the continuous normal bone development in the unaffected areas result in marked deformity and shortening of the affected extremity.

Chronic Idiopathic Hypertrophic Osteo-Arthropathy.

JOHN D. CAMP AND R. L. SCANLAN (*Radiology*, May, 1948) state that chronic idiopathic hypertrophic osteo-arthropathy is a condition occurring predominantly in males at the age of puberty or adolescence and characterized by the osteo-arthropathic syndrome—clubbing of the digits, enlargement of bones and joints, and thickening of the skin of the face—in the absence of any demonstrable primary disease. Its course is slowly progressive to deformity and disability, which are often of extreme degree. Radiography is often of indispensable aid, not only in distinguishing osteo-arthropathy, but in differentiation of the type. The diagnostic basis is the characteristic periosteal formation of new bone, as related to distribution, *tempo* and duration of the disease process, as manifested in the radiograms, coupled with the absence of evidence of disease in other parts of the body. Of the systemic diseases capable of causing confusion, acromegaly has been, at least historically, the most important. Radiographically, acromegaly is almost the exact antithesis of chronic osteo-arthropathy. In acromegaly the short tubular bones are increased in length, as a rule with proportionately slender diaphyses and hypertrophied cancellous extremities. The ungual tufts are hypertrophic, and the sites of tendon and ligamentous insertions are also hypertrophied. Degenerative joint changes are the rule in advanced cases, with resultant hypertrophic changes. All these features are conspicuously absent in osteo-arthropathy, the opposite changes prevailing, namely, normal bone length and normal metaphyses, thickened diaphyses, atrophic ungual tufts and normal epiphyses and joints. Periostitis, specific and non-specific, offers difficulty only on single films. Syphilitic periostitis lacks the characteristic symmetrical distribution; for example, it occurs usually on tibiæ, not simultaneously on fibulæ, and seldom on the bones of the forearm. It is usually not concentric. Non-specific periostitis, as, for example, that associated with infections of the foot in diabetic patients, shows soft tissue change and destruction of bone as well as the periosteal proliferation, and in addition there is usually endosteal proliferation, with narrowing of the medullary canal. Advanced *osteitis deformans* may be similar in some respects to advanced idiopathic osteo-arthropathy. The age of onset, presence of cystic changes or bowing deformities, distribution of involved bones, and disordered trabeculation with involvement of the medullary canal should lead to easy distinction. Osteopetrosis shows narrowing of the medullary canal and is thus readily distinguished. Of the other types of osteo-arthropathy the hereditary form may be difficult to differentiate after puberty. Secondary osteo-arthropathy is rarely difficult to distinguish and then only after puberty; since the syndrome is most often acute in onset, is rapid in *tempo*, lasts months rather than years, and is associated with systemic disease, osteoporosis due to disuse accompanies the painful joints and is revealed in both the new and the old bone, the new bone usually being deposited as a single wide layer. The ungual tufts may or may not be absorbed. If the primary disease is chronic, slowly progressive and of long duration, it may produce changes identical with those of the chronic idiopathic variety. A few conclusions may be drawn as regards the relationship of the chronic idiopathic type to other types of osteo-arthropathy. First, osteo-arthropathy can occur in the absence of primary disease or of Mendelian dominant heredity; as a matter of fact, it can occur in its most extreme form in the absence of these conditions. Secondly, onset at puberty in these cases reveals close relation to the glands of internal secretion, particularly the pituitary. Thirdly, there would appear to be a central initiating factor, possibly pituitary or hypothalamic or both, and a peripheral effector mechanism, probably circulatory, and capable in certain instances of causing secondary clubbing and other characteristic changes in a single digit or extremity. The final factor, and perhaps the greatest in importance, is the constitutional predisposition which accounts for familial tendencies and variance in individual susceptibility to osteo-arthropathy.

PHYSICAL THERAPY.

Medulloblastoma.

C. B. PIERCE, W. V. CONE, J. BOUCHARD AND R. C. LEWIS (*Radiology*, May, 1949) state that medulloblastomata are sensitive to irradiation, but in a large number of cases the disease is advanced by the time a diagnosis is made, so that the affected child's condition is poor. The operative mortality rate is high, averaging about 20%, and the authors considered that, if surgical intervention could be avoided, this operative mortality would be eliminated, and the

results improved. Accordingly they performed aspiration biopsy on tumours which appeared clinically to be medulloblastoma, following it by X-ray therapy when the diagnosis was established. They do not agree that X-ray therapy of suspected cerebellar tumours should be given without histological confirmation, on the grounds that time would be lost in such conditions as abscess or cystic astrocytoma in which the method of choice is operation. They state that aspiration biopsy is an easy method of obtaining tissue from the tumour, but is carried out only if the intracranial tension is high, to avoid possible complications from a ruptured vessel. Radiation therapy can be commenced within a few hours of the patient's admission to hospital with the authors' technique, in contrast to a delay of a week or ten days with suboccipital craniotomy. The authors consider that the field of irradiation should include the upper half of the cervical part of the spinal cord and that a tumour dose of not less than 4500*r* should be delivered in about four weeks. Five patients in whom the disease was very advanced have been treated in this manner. Within three days of commencement of irradiation, improvement was striking in three cases, and on completion of the course, all patients had lost the majority of their symptoms. The number of cases is too small to offer more than a preliminary report, but the patients were all "poor surgical risks" and the immediate results of irradiation have been striking.

X-Ray Therapy for Pituitary Adenoma.

A. BACHMAN AND WILLIAM HARRIS (*Radiology*, September, 1949) state that pituitary adenomata frequently respond to radiotherapy, but review of the literature shows that the methods employed and the results vary widely. The authors have a series of 64 cases in which treatment was carried out by varying techniques; they have analysed the results in order to determine which method was the most effective. Radiotherapy was the primary treatment in 61 of the 64 cases. Three patients were first treated surgically and given irradiation for recurrence; thirteen patients had surgery following irradiation. Techniques varied in the total dose delivered, the number of courses given, the treatment period for each course, and the over-all time of treatment. The physical factors of treatment were uniform. Four types of result were found: (i) great improvement (patients restored almost to normal), (ii) moderate improvement, (iii) doubtful, and (iv) no effect. It was found that the basophile adenoma yielded a higher proportion of results classed as great improvement than the other types, whatever technique was employed. The most important findings were in the tables correlating results with dosage. The best results were obtained with patients receiving a tumour dose of between 2000*r* and 3000*r*. The number who received over 3000*r* was too small to allow conclusions to be drawn. With doses below 2000*r* the results were inferior. Multiple-course techniques with comparatively small doses per course are commonly used in America, but it was found that the results in the second and later courses were much inferior to those in the first. Patients who showed a satisfactory result with repeated courses received an average total dose of 4070*r* compared with a total average dose of 2290*r* amongst those yielding a satisfactory response to a single course. In addition, with a single course, improvement occurred earlier. Of the 31 patients whose results were satisfactory, five have had recurrence of symptoms in between six months and three years. The authors state that, according to the literature, the best results appear to have been obtained by Kerr, who reports 70% of good results in 50 patients, with a single series technique of 3000*r* tumour dose in one month; this appears to be the optimal dosage. Of pituitary adenomata, 20% are cystic and 10% are radioresistant, so that results better than 70% by radiotherapy cannot be expected. The important points stressed are that tumour doses below 1000*r* are inadequate, even if repeated, and that if the initial course of treatment adequately given is ineffective, a further course is unlikely to produce results; that is, the tumour is radioresistant or cystic. Cases in which further improvement follows a second course of treatment indicate that a higher dosage in the first instance might have produced better results. The authors consider that in the average case a tumour dose of 3000*r* to 4000*r* delivered in thirty to forty-five days is the optimum. If this fails, surgery seems indicated. In the advanced cases with bone destruction, higher dosage to 6000*r* can be considered, as in these cases poor results are obtained from surgery and the condition may even be inoperable. Cushing's series showed a five-year recurrence-free rate of 57·5% with surgery alone; with post-operative irradiation, this was raised to 87%, and in some instances the amount of irradiation would now be regarded as too small.

Supervoltage X-Ray Therapy.

S. T. CANTRIL AND F. BUSCHKE (*Radiology*, September, 1949) present an appraisal of the usefulness of supervoltage X-ray therapy as judged by experience at The Swedish Hospital over a period of thirteen years. The equipment used was a General Electric KXC—2, with an operational voltage of 800 kilovolts, a half-value layer equivalent of 9·1 millimetres of copper or 3·0 millimetres of lead. The authors state that the physical advantages are an improved depth dose, although this is relatively small, and better definition of the beam due to greatly reduced scatter in tissue. This allows for greater efficiency in treatment as measured by the relative proportion of energy absorbed by the tumour to the total energy absorption. Owing to these factors, irradiation sickness is more rare and easier to control with the higher voltage range. Another advantage is lessened skin damage with 800 kilovolts as compared with 200 kilovolts. The combination of greater penetration and less skin damage allows for treatment of certain lesions through one field. There are certainly disadvantages, compared with the use of a 200 kilovolt apparatus, mainly cost and loss of flexibility. The authors point out that with supervoltage X-ray therapy, no startling improvement could be anticipated in tumours which did not respond to medium voltage therapy, for example, fibrosarcoma, osteogenic sarcoma or melanoma. The intestines do not tolerate a higher dose of supervoltage X rays than that of those in the range of 200 kilovolts, and hence the limitation to effective pelvic irradiation is still present. Ischæmia and previous irradiation are still deterrents to effective therapy. The most that can be expected from radiation of higher voltage is improvement in results for those forms which are essentially responsive to medium voltage therapy. For discussion there are two groups, deep-seated and accessible tumours. In the first group, the most important are tumours of the urinary bladder, *cervix uteri*, œsophagus and pituitary. For the bladder, a tumour dose of 5000*r* to 6000*r* is given over six weeks; it is noted that the skin is no guide to dosage. The most vulnerable tissue in the field is the rectum, and with too high a dose fibrosis will occur. A similar problem occurs with carcinoma of the uterine cervix. A combined intracavitary and external irradiation technique is used. The primary disease is controlled in almost every case, but extracervical spread presents difficulties which supervoltage X-ray therapy has not solved. It is estimated that the cervix may safely receive 20,000*r*, the underlying rectum 5000*r*, the paravesical tissues 8000*r*, and the intestines within the pelvis 4500*r* to 5000*r* only. With 800 kilovolts two anterior and two posterior portals with a central gap over the cervix and rectum deliver 3000*r* to the parametrium in three weeks. This combined with 1800*r* to 2000*r* from radium is regarded as the limit of safety. It has been previously reported that, in the early days of supervoltage therapy, intestinal necrosis occurred before the depth dose potentialities of supervoltage irradiation were realized. The skin reaction associated with this parametrial dose rarely reaches the stage of moist desquamation. The general constitutional disturbance, however, is minimal. Results achieved are not superior to those of the Institut de Radium, with a comparable radium technique and X-ray therapy with a voltage of 200 kilovolts. The reason for failure is not the wavelength, but the vulnerability of normal tissue in the pelvis, and it is difficult to see how X rays generated at even 2000 kilovolts can improve results. In treatment of carcinoma of the œsophagus, the picture is more hopeful. With 200 kilovolts it is rarely possible to give a tumour dose over 4000*r*. With 800 kilovolts a tumour dose of 5000*r* to 6000*r* can be given in the same period, and general disturbances are minimal, a fact which is important for patients in poor general condition. In treatment of accessible cancers, several advantages are present with supervoltage X-ray therapy. The beam is well localized, with a sufficiently low skin effect to permit irradiation through a single field if necessary. A disadvantage may be the irradiation of tissue beyond the tumour, although this may be less than that with 200 kilovolts and multiple fields. Several factors must be considered in the choice of technique. A warning is given of the danger of causing necrosis of subcutaneous tissue and bone by a dose which the skin will readily tolerate. With the possible exception of carcinoma of the base of the tongue, tumours of the head and neck yielded with supervoltage X-ray therapy results no better than those with 200 kilovolts.

British Medical Association News.

ANNUAL MEETING.

THE annual meeting of the Queensland Branch of the British Medical Association was held at the Medical School, Brisbane, on December 9, 1949, DR. B. L. W. CLARKE, the President, in the chair.

The President welcomed to the meeting Dr. J. G. Hunter, General Secretary of the Federal Council, and Dr. Mervyn Archdall, Editor of THE MEDICAL JOURNAL OF AUSTRALIA. Both visitors subsequently addressed the meeting.

ANNUAL REPORT OF THE COUNCIL.

The annual report of the Council which had previously been circulated among members was taken as read and adopted on the motion of Dr. H. W. Horn, seconded by Dr. Norman Sherwood. The report is as follows.

The Council has pleasure in presenting the fifty-fifth annual report of the work of the Branch for the year ending November 15, 1949.

Membership.

The membership of the Branch is 798, *plus* 4 honorary members as against 732, 1 complimentary and 4 honorary members in 1948, making a total gain of 65. There are also 178 honorary associate members, 78 of whom were elected this year.

The gains were: new members 54, transfers from other Branches 58, members reinstated 1, members reelected 3.

The losses were: members transferred from Branch 39, resignations 5, deceased 4, struck off 3.

Obituary.

We regret to record the deaths of the following members: Dr. F. R. Benson, Brisbane; Dr. Clive Sippe, Brisbane; Dr. H. J. Stewart, Brisbane; Dr. Hilda Wienholt, Brisbane; Dr. J. H. Crawford, junior, Pomona.

Meetings.

In addition to the annual meeting, ten general meetings of the Branch were held, including two clinical meetings; a special general meeting was held to discuss *The National Health Service Act* and amendments to *The Pharmaceutical Benefits Act*. The average attendance at the ordinary meetings was 46, and 422 attended the special general meeting.

Council.

Twenty-one meetings of the Council were held. Record of attendance is as follows:

Dr. B. L. W. Clarke (President)	18
Dr. H. R. Love (President-Elect)	20
Dr. Norman Sherwood (Past President)	13
Dr. H. W. Horn (Honorary Treasurer, Federal Council Representative, Chairman of Committees)	20
Dr. J. R. Adam (Honorary Secretary)	21
Dr. E. Lorimer Walker (Honorary Secretary of Committees)	20
Dr. Felix Arden (Councillor)	15
Dr. Ronald S. Bennett (Councillor)	17
Dr. John W. Best (Councillor)	12
Dr. A. W. Eklund (Councillor)	17
Dr. K. B. Fraser (Councillor)	15
Dr. Glen V. Hickey (Councillor)	16
Dr. Alan E. Lee (Federal Council Representative, Councillor)	18
Dr. A. D. A. Mayes (Councillor)	21
Dr. H. S. Patterson (Councillor)	19
Dr. Athol Quayle (Councillor)	15
Dr. R. G. Quinn (Councillor)	12
Dr. Arnold Robertson (Councillor)	16

Scientific and Medico-Political.

February.—Clinical meeting in conjunction with the Brisbane Hospital Clinical Society.

March.—Dr. I. M. Mackerras: "The Prevention of Gastro-Enteritis in Infants."

April.—Dr. A. R. Murray: "Traumatic Surgery."

May.—Symposium: "The Use of Anticoagulants." Dr. Noel Gutteridge, Dr. Peter Row and Dr. F. J. Booth.

June.—Sir Henry Newland: "Stages in the Development of the Operative Surgery of Prostatic Obstruction" (The Bancroft Oration).

August.—Professor W. V. Macfarlane: Physiology demonstration.

September.—Dr. R. A. O'Brien: "Medical History—Do We Learn Enough from It?" (The Jackson Lecture).

October.—Dr. R. G. Quinn: "The Scope of General Practice."

November.—Clinical meeting in conjunction with the Mater Misericordiæ Hospital Clinical Society.

The July meeting was cancelled owing to the lighting restrictions during the coal strike.

Office Bearers and Councillors.

Dr. Harold R. Love was elected President-Elect for the year 1949. Dr. J. R. Adam was elected to the position of Honorary Secretary.

The following office bearers were elected by the Council:

Honorary Treasurer.—Dr. H. W. Horn (reelected).

Chairman of Committees.—Dr. H. W. Horn (reelected).

Honorary Secretary of Committees.—Dr. E. Lorimer Walker.

Honorary Librarian.—Professor Neville G. Sutton (reelected).

Assistant Honorary Librarian.—Dr. Konrad Hirschfeld (reelected).

Council.—The following members are not seeking reelection for the ensuing year: Dr. Norman Sherwood (Past President), to whom the Council and members owe a debt of gratitude for the many years of valuable service he has given to the work of the Branch. Dr. R. G. Quinn is another Councillor who will be greatly missed, and Dr. John W. Best (Warwick) and Dr. A. W. Eklund (Nambour), who have travelled many hundreds of miles during the year to attend fortnightly meetings of the Council, and Dr. Athol Quayle.

Ethics Committee.

At the annual meeting of the Branch held on December 10, 1948, the following were reelected members of the Ethics Committee: Dr. J. G. Avery, Dr. Val. McDowall, Dr. L. J. J. Nye, Dr. J. J. Power, Dr. M. Graham Sutton, Dr. J. G. Wagner. Dr. F. W. R. Lukin was elected to fill a vacancy caused by the death of the late Dr. D. Gifford Croll. Three meetings of the Ethics Committee have taken place during the year, and at the first of these Dr. M. Graham Sutton was appointed Chairman and Dr. F. W. R. Lukin, Honorary Secretary. Owing to his departure from the State, Dr. Avery tendered his resignation as a member of the committee in August, which was accepted with regret by the Council.

Joseph Bancroft Oration.

The twenty-fourth Bancroft Oration was delivered by Sir Henry Newland, C.B.E., D.S.O., on Friday, 3rd June, 1949, at the Lecture Theatre of the Medical School. The title of the oration was: "Stages in the Development of the Operative Surgery of Prostatic Obstruction."

A vote of thanks was moved by Dr. F. W. R. Lukin, seconded by Dr. Alan E. Lee, and carried by acclamation. At the conclusion of the oration the President presented the Bancroft Memorial Medal to Sir Henry Newland.

Jackson Lecture.

The annual lecture in memory of the late Ernest Sandford Jackson was delivered by Dr. R. A. O'Brien, O.B.E., M.D., D.P.H., R.C.P. and S., who chose for his subject "Medical History—Do We Learn Enough from It?". The lecture took place on Friday, 2nd September, 1949, at the Lecture Theatre of the Medical School, University of Queensland.

Library.

The facilities provided by the library are appreciated by members, and during the year 142 books were borrowed by 67 members, which number includes country members who have written requesting that books be sent under the existing conditions that they defray cost of postage. An order has been placed with the publishers of the following journals: *Pediatrics* and *Acta Otolaryngologica*.

Dr. G. A. C. Douglas has donated to the library complete volumes of *The Journal of Bone and Joint Surgery* from 1934 to 1947.

Representation.

The Branch was represented as follows during the year:

Council of the British Medical Association.—Dr. Isaac Jones.

British Medical Association Representative Meeting, Harrowgate, 1949.—Dr. J. Lloyd Simmonds.

Federal Council of the British Medical Association in Australia.—Dr. H. W. Horn, Dr. A. E. Lee.

Australasian Medical Publishing Company, Limited.—Dr. Alan E. Lee (director), Dr. T. A. Price, Dr. H. W. Horn (members).

Medical Assessment Tribunal.—Dr. Alex. Marks.

Queensland Medical Board.—Dr. R. G. Quinn, Dr. J. G. Wagner, Dr. F. W. R. Lukin.

Medical Officers' Relief Fund (Federal).—Queensland Committee: Dr. W. H. Steel, Dr. G. W. Macartney, Dr. K. B. Fraser.

Post-Graduate Medical Education Committee.—Dr. A. E. Lee, Dr. Felix Arden, Dr. Harold R. Love, Dr. J. R. Adam.

Queensland Institute of Medical Research.—Dr. W. H. Steel.

Queensland Bush Nursing Association.—Dr. L. Bedford Elwell.

Queensland Council of Social Agencies.—Dr. G. B. V. Murphy.

Flying Doctor Service of Australia.—Dr. Harold Crawford.

Red Cross Blood Transfusion Service.—Dr. Milton Geaney.

The Surf Life Saving Association of Australia (Queensland Centre).—Dr. F. W. R. Lukin (liaison officer).

National Safety Council of Australia (Queensland Division).—Dr. L. A. Little.

Physical Fitness Association of Queensland.—Dr. E. S. Meyers, Dr. Harold Crawford.

Australian Association for Better Hearing.—Dr. Herbert Earnshaw, Dr. J. R. Hutcheon.

Queensland Bush Children's Health Scheme.—Dr. Felix Arden.

Board of Studies in Physiotherapy.—Mr. J. R. S. Lahz.

Federal Medical War Relief Fund: Local Committee of Management.—Dr. J. G. Wagner (chairman), Dr. F. W. R. Lukin (honorary secretary), Dr. M. Geaney, Dr. J. G. Avery, Dr. J. V. Duhig.

Queensland Health Education Council.—Dr. Felix Arden.

Florence Nightingale Memorial Committee of Australia (Queensland Branch).—Dr. Norman Sherwood.

The Editor of THE MEDICAL JOURNAL OF AUSTRALIA was represented by Dr. Felix Arden.

Organization Subcommittee.

Personnel: Dr. H. W. Horn (chairman), Dr. Alan E. Lee, Dr. Arnold W. Robertson, Dr. K. B. Fraser, Dr. R. G. Quinn, Dr. A. D. A. Mayes, Dr. Athol Quayle, Dr. R. S. Bennett, and the ex-officio members of the Council.

Twenty-two meetings of the subcommittee were held, and, as usual, dealt with many matters and submitted recommendations to the Council where policy was involved.

By-Laws.

Amendment of By-Law 4.—At the last annual meeting of the Branch held on December 10, 1948, it was unanimously resolved that the annual subscription of members be as follows: senior metropolitan members £9 9s., senior country members £8 8s., metropolitan professional officers £7 7s., country professional officers £6 6s., university staff £6 6s., junior members (under three years' qualification) £4 4s., retired members and members over seventy years of age £4 4s.

The Council has ruled that, provided a medical superintendent of a public hospital has no right of private practice, the rate payable by him shall be that of a professional officer, as provided in the amended by-law. Medical registrars of the Brisbane Hospital (teachers) will be charged the same rate of subscription payable by the university staff.

Government Medical Officers.

Repatriation Medical Officers' Association.

Assistance has been given to the Repatriation Medical Officers' Association in its approach to the Public Service Arbitrator regarding the conditions of service and salaries of medical officers employed by the Repatriation Department, and evidence in support of their claim has been taken from Queensland members.

Salaried Medical Officers.

The Federal Council has appointed a committee consisting of a representative from each State to report on desirable salary standards for all classes of full-time medical appointments Commonwealth and State. Dr. H. W. Horn is the Queensland representative.

All available information is being collected and will be further discussed and collated, and the Council hopes that this will result in a big improvement in the conditions of government medical officers in this State.

Public Health.

City Medical Officer of Health, Brisbane.—Dr. Alan Ashworth, who will arrive in Brisbane in November, has accepted appointment to this position and his membership has been transferred from the London Branch of the British Medical Association to the Queensland Branch.

Poisons Regulations.—The attention of members has been drawn to their responsibility under the Poisons Regulations when prescribing dangerous or restricted drugs for private patients, both in their homes and in hospitals, and they have been advised to conform strictly to the Act in this regard.

Lodges.

The capitation fee for the metropolitan area computed by the Government Statistician's office to come into operation as from July 1, 1949, was 41s. 6d.; the income limit for the same period, which now also varies in accordance with the Nominal Wage Index Figure, was as follows: new members £489 *per annum*, present members £652 *per annum*.

During the year many more country lodge medical officers have made agreements with their lodges in conformity with the metropolitan agreement, that is, on sliding scale for fixation of capitation rate and income limit.

Informal discussions are taking place with the Queensland Friendly Societies Association to consider some necessary extensions of the lodge service and financial cover for this additional service.

Liquid Fuel Subcommittee.

Personnel.—Dr. K. B. Fraser, Dr. E. Lorimer Walker, Mr. F. K. Davis, secretary. This committee, which had been disbanded when petrol rationing was abolished, has now been reconstituted. The Liquid Fuel Board has requested the British Medical Association, as in the past, to deal with the requirements of metropolitan doctors.

Memorial Roll, Queensland Branch of the British Medical Association.

The name of David Gifford Croll has been added to the roll.

The Memorial Roll comprises a handsomely bound volume; each page commemorating a member has an inscription which is hand illuminated and engraved.

Affiliated Local Associations.

The annual conference of delegates of affiliated local associations of members with the Council was held on Wednesday, June 1, 1949, at which the following were represented: Downs and South-Western, Dr. M. Graham Wilson; Ipswich and West Moreton, Dr. M. A. Williams; Rockhampton, Dr. E. R. Watkins; Cairns, Dr. J. Brody; Townsville, Dr. G. H. Moore; Mackay, Dr. Robert M. Grant and Dr. J. G. Morris; South Coast, Dr. O. N. Lloyd; Nambour, Dr. J. E. Trotter; Warwick, Dr. W. G. Oakeley. In addition, Dr. L. P. Winterbotham and Dr. Garth May represented the General Practitioner Group.

Apologies were received from the Gladstone and Bundaberg local associations who were unable to send delegates.

Matters of mutual and local interest were discussed, and at the conclusion of the meeting visiting delegates were entertained at luncheon by members of the Council.

It is felt that this annual gathering of country members provides a valuable opportunity of discussing matters at first hand with the Council, and thereby bringing about a better understanding of the problems of each which cannot always be conveyed by correspondence.

Representatives of local associations also attended the special meeting of the Branch held on March 22 to consider the national health service and amendments to *The Pharmaceutical Benefits Act.*

There are now thirteen affiliated local associations, the latest formed being Gladstone and South Coast.

The Council appreciated very much the help given by the local associations in distributing locally publicity matter, which expedited delivery.

No annual reports have been received from the following local associations: Cairns, Lower Burdekin (Ayr), North Coast (Gympie) and Maryborough.

Rockhampton Local Medical Association.

Our association has been very active during the past year. The Post-Graduate Medical Education Committee may be assured that the visits of medical men on the northern lecture tours are very much appreciated by this association—the lecturers choosing subjects of everyday interest.

Again our thanks are due to the Rockhampton Hospital medical superintendent for his organization of the clinical meetings which have been reasonably well attended.

We have, during the year, had two or three association dinners, which proved to be an admirable means of getting together the local and district medical men for an informal chat and mellow deliberation.

Office Bearers.—Dr. F. C. Wooster was reelected President and Dr. E. R. Watkins Honorary Secretary. The membership is 29.

Inquiry from pharmacists has shown that not one Commonwealth form for free medicine has been issued by any local practitioner. The response to the Federal Independence Fund was excellent—constituting what was considered to be about 90% of the total suggested for our members.

E. R. WATKINS,
Honorary Secretary.

Townsville Local Medical Association.

The following is the report of the activities of the Townsville Local Association for the year ending October, 1949.

Great interest has been shown by members of the association in both clinical and medico-political affairs. Meetings were held fortnightly throughout the year, programmes being so arranged that clinical meetings alternated with general business and medico-political sessions. Several special meetings were held in regard to *The Pharmaceutical Benefits Act*. Dr. P. H. Monahan represented the Townsville Local Association at a special meeting called by the State Council on March 2 in connexion with *The Pharmaceutical Benefits Act*, and Dr. G. H. Moore was the representative at the local associations' conference with the Branch Council in Brisbane in June.

In the medico-political field the members of this association have presented a solid front of opposition to *The Pharmaceutical Benefits Act* and the *National Health Service Act*.

Attendances have been satisfactory at all meetings, particularly the numbers of younger members. The membership is now 19, one of the older practitioners having retired.

A library has been started by members and promises to offer a very useful variety of medical and surgical literature.

The annual general meeting was held on February 17, 1949, when Dr. L. Halberstater was elected President and Dr. G. H. Moore Secretary-Treasurer. The programme committee for the year consisted of the President, Secretary, Dr. T. U. Ley and Dr. P. J. Monahan.

The Post-Graduate Medical Education Committee sent visiting lecturers on two occasions and the lectures and clinical demonstrations were greatly appreciated. Professor Shedden Adam visited Townsville in July and Dr. Ellis Murphy and Dr. Alan Lee in September. The local association was also privileged to have as a guest and lecturer in July Sir Carrick Robertson, of Auckland, New Zealand.

At the conclusion of each programme the association entertained the guests and visitors at a most enjoyable dinner. The country practitioners showed great interest in these lecture tours and their attendance is very pleasing, as they have long distances to travel to Townsville.

Clinical meetings held were as follows:

March.—Clinical cases presented by Dr. Monahan and Dr. Joyce.

April.—Showing of films from the Post-Graduate Medical Film Library: 1. "Technique of Skin Grafting." 2. "Time Factor in the Treatment of Burns." 3. "Open Drop Ether Anæsthetics."

May.—Clinical cases presented by Dr. White, Dr. King and Dr. Ellis.

June.—Paper presented by Dr. de Salis: "Laboratory Findings in Common Blood Disorders."

July.—Showing of films by courtesy of Taylors and Elliotts Proprietary, Limited: 1. "Treatment of Varicose Veins." 2. "Treatment of Fractures of the Upper Limb."

August.—Paper presented by Dr. T. U. Ley on "Common Foot Disabilities". Clinical cases presented by Dr. King, Dr. Rothfield and Dr. Luke.

L. HALBERSTATER,
President.

Mackay Local Medical Association.

Quarterly meetings of the Mackay Local Medical Association were held during the year, each meeting having a good attendance. An extraordinary general meeting was held on March 24, 1949, to receive a report from our representative, Dr. Ian Chenoweth, on the activities of the Branch meeting held in Brisbane on March 22 concerning *The Pharmaceutical Benefits Act* and the National Health Service Bill.

An attempt has been made to replace honorary service by local practitioners at the Mackay General Hospital with a paid part-time service. Negotiations have been started by our local association.

Mackay has closely followed the negotiations concerning the proposed *Pharmaceutical Benefits Act* and the National Health Service Bill.

Association with the Mackay and District Friendly Societies continues on a friendly basis. The lodge capitation fee for 1949-1950 has been agreed upon at 41s. 6d.

A scheme whereby Mackay practitioners may obtain annual holidays, by a pool system of locums, is under consideration.

Office Bearers.—The office bearers elected at the annual meeting are: President, Dr. P. W. Hopkins; Honorary Secretary-Treasurer, Dr. S. C. Williams.

Lectures.—Lectures given during the year at the quarterly meetings were: Dr. H. J. Taylor, "Glaucoma"; Dr. G. Candi, "Convalescence of Acute Rheumatic Fever and the Value of Blood Sedimentation Rate"; Dr. S. C. Williams, "Tendon Injuries of the Hand"; Dr. K. Whitehead, "Low Back Pain and Sciatica". Visiting lecturers during the year have been Dr. H. R. Love, Dr. A. J. Foote, Professor G. Shedden Adam, Dr. Alan Lee and Dr. Ellis Murphy. Our thanks are expressed for these post-graduate visitors.

S. C. WILLIAMS,
Honorary Secretary.

Nambour District Local Medical Association.

The year 1948-1949 has not been as successful as we would have hoped, but we look forward to greater success in the future. This is probably owing to the fact that members are all general practitioners in a widely scattered area and consequently find it difficult to congregate regularly for general discussion, lectures *et cetera*.

The annual general meeting was, perhaps, the best attended throughout the year, and Dr. Kesteven, who recently arrived in the district from Cooktown, was elected President, the position of Secretary-Treasurer being filled by Dr. J. E. Trotter.

In spite of the fact that we have not met often, we meet each other frequently and freely discuss our problems, which in itself has kept the local association going. Several film evenings have been held, and lecturers have visited us from Brisbane.

We have an extensive programme of films and lectures which we hope to hold during the ensuing twelve months. During the past year we were unfortunate in losing three active members, two of whom are resident in Sydney and the third transferred to Thursday Island. In their place we welcomed Dr. Toyne and Dr. Kesteven, both keen and interested persons, who we hope will help to foster our local association.

J. E. TROTTER,
Honorary Secretary.

Bundaberg Local Medical Association.

During the past year the Bundaberg Local Medical Association has held nine meetings, and the membership has increased to nine. The office bearers elected are: President, Dr. Egmont Schmidt; Honorary Secretary, Dr. Eric Schmidt.

Most of our meetings concerned the *National Health Service Act* and *The Pharmaceutical Benefits Act*. Dr. A. W. Graham represented the association at the special meeting in Brisbane to discuss these matters. Throughout we have been wholeheartedly behind our Federal Council in all they have done.

Recently the Post-Graduate Medical Education Committee agreed to send visiting lecturers to Bundaberg, and we are looking forward in the near future to lectures in pædiatrics, obstetrics and gynæcology.

ERIC E. SCHMIDT,
Honorary Secretary.

Downs and South-Western Local Medical Association.

The annual meeting of the Downs and South-Western Local Medical Association was held in Toowoomba on September 17, 1949. The following office bearers were elected: President, Dr. A. W. L. Row; President-Elect, Dr. W. F. Machin; Honorary Secretary-Treasurer, Dr. D. F. Farmer.

During the year five lectures were given at general meetings, namely: Dr. Harold Love, "High Blood Pressure"; Dr. J. G. M. Beale, "The Basis of Radiological Interpretation in Diseases of the Chest"; Dr. A. Fryberg, "Poliomyelitis"; Dr. F. W. R. Lukin, "The Present Position in Prostatic Surgery"; Dr. K. B. Fraser, "Some Aspects of Treatment of

Cleft Palate" (in conjunction with the Toowoomba Branch of the Australian Dental Association).

We are grateful to the four lecturers who visited us from Brisbane, and each one of them added to our fund of knowledge in some particular aspect. Our thanks are also due to Dr. Beale, for his lecture, which had been most carefully prepared, was excellently presented, and was very much appreciated by all members present.

An enjoyable clinical evening was held on November 13, 1948, and cases were presented by Dr. Furness, Dr. Hickey, Dr. Spark, Dr. Deithe, Dr. Row and Dr. Graham Wilson.

Four executive meetings were held during the year in October and December, 1948, and April and June, 1949, but attendance at these meetings was not as good as it should have been.

Official Representation.—During the year the association was represented at the annual dinner of the Toowoomba Branch of the Australian Dental Association. Six members attended a general meeting of the Queensland Branch of the British Medical Association in Brisbane with regard to *The Pharmaceutical Benefits Act* and the *National Health Service Act*. The medical profession in Queensland was extremely well represented at this very large meeting of medical men, and all those present were given much food for thought. The honorary secretary attended a general meeting of the New South Wales Branch in Sydney in April in connexion with the abovementioned Acts, and represented the association at the annual conference of the local medical associations with the Queensland Branch Council in Brisbane in June.

Membership.—The membership of the association at present stands at 37 financial members, and our finances are in quite a satisfactory position.

Acknowledgements.—Our thanks are due to the Toowoomba Hospitals Board and the medical superintendent for making their board room available for our meetings, and to the matron of the Toowoomba General Hospital for allowing us to use the nurses' lecture room for Dr. Fraser's lecture. Also, our thanks go to Dr. Row for his skilled assistance and technical knowledge in the use of the epidiascope, and to Dr. Hickey for his liaison, in many useful ways, with the Branch Council.

M. GRAHAM WILSON, President.
J. W. P. HENDERSON, Honorary Secretary.

South Burnett Local Medical Association.

The following is a report of the association's activities for the year ending September 30, 1949.

Office Bearers.—President, Dr. G. Ruscoe; Honorary Secretary, Dr. C. H. Wood.

The membership of the association is eight, comprised of medical practitioners from Kingaroy, Nanango, Wondai, Murgon and Goomeri, and one life member.

Dr. R. J. Nash was elected a life member of the association in view of his long service in the district and to the association. We regret his departure from the area as a result of ill health.

A meeting of the association was held in Kingaroy in November, 1948, when Dr. P. H. Macindoe addressed members on "Plastic Surgery and Repair", and in August, 1949, a lecture was given at Murgon by Dr. S. C. Suggit on "Common E.N.T. Ailments". We are grateful to the Post-Graduate Medical Education Committee for arranging these visits.

CHARLES H. WOOD,
Honorary Secretary.

South Coast Local Medical Association.

The South Coast Local Medical Association came into being on April 21, 1949, when the inaugural meeting was held and the following officers were elected: President, Dr. W. A. Mackey; Vice-President, Dr. W. J. Matthews; Honorary Secretary, Dr. T. H. Gaven; Honorary Treasurer, Dr. O. N. Lloyd; Honorary Auditor, Dr. W. H. Nette. The membership is eleven.

Regular meetings have been held. One visiting lecturer, Dr. Felix Arden, addressed members on "Fat Intolerance and Allied Disorders"; in the coming year it is intended to have as many visiting lecturers as possible to visit us.

At the annual general meeting held on September 29, 1949, the following officers were elected: President, Dr. E. Culpin; Vice-President, Dr. W. J. Matthews; Honorary Secretary, Dr. O. N. Lloyd; Honorary Treasurer, Dr. W. H. Nette; Honorary Auditor, Dr. R. Levy.

T. H. GAVEN,
Honorary Secretary.

Ipswich and West Moreton Local Medical Association.

The Ipswich and West Moreton Local Association, now in the twenty-third year of its existence, has at present 24 members, comprising 16 town and 8 country members.

Visiting lectures, arranged through the Post-Graduate Medical Education Committee, were: Dr. David Jackson, Dr. A. J. Foote, Dr. H. Masel, Dr. P. H. Macindoe, Dr. Hugh McLelland, Dr. L. W. Gall, Dr. B. L. W. Clarke and Dr. T. V. Stubbs Brown. In addition, on October 21, 1948, Dr. H. G. Wilson, Dr. D. A. Cameron, Dr. G. C. Wilson, Dr. D. A. Carter and Dr. H. S. Patterson showed a series of cases at a clinical meeting.

At the annual general meeting on March 17, 1949, Dr. M. A. Williams was elected President of the association for the ensuing year and Dr. H. S. Patterson Honorary Secretary. The President and Secretary represented this association at the meeting with the Council of the Queensland Branch of the British Medical Association held during post-graduate week. The annual dinner was held at the Hotel Grande and members of the local Dental Association were guests at this dinner.

The happy spirit of good fellowship which has always been a feature of this association, and which was in no small measure owing to the influence of the late Dr. J. A. Cameron, has continued, and is frequently remarked upon by visiting lecturers.

H. S. PATTERSON,
Honorary Secretary.

Gladstone and District Local Association.

Two meetings only were held during the year owing to the difficulty in getting members together at the one time. Dr. C. Kingston resigned from membership as he was leaving the district to take up practice at Pittsworth. He was succeeded as medical superintendent at the Gladstone General Hospital by Dr. W. H. M. Fraser, who became a new member of the association.

At the annual meeting of the local association held on October 26, 1949, Dr. J. A. McGree was elected President and Dr. W. H. M. Fraser Honorary Secretary-Treasurer.

Negotiations are still proceeding with the local friendly societies to have the lodge capitation fee brought up to the same level as the Brisbane metropolitan rate and to ensure stricter supervision of the upper income limit of proposed new members.

W. H. M. FRASER,
Honorary Secretary.

Sections for Study of Special Branches of Medical Knowledge.

Anæsthesia.—Dr. Arnold Robertson, Queensland State Representative.

Medical Historical.—Dr. John Bostock, Chairman; Dr. G. B. Murphy, Honorary Secretary.

Obstetrical and Gynæcological.—Dr. Robin Charlton, President; Dr. Roy Hemsley, Honorary Secretary.

Ophthalmological.—Dr. F. Garret Scoles, President; Dr. James Hart, Honorary Secretary.

Orthopædics.—Dr. George Douglas, Chairman; Mr. J. R. S. Lahz, Honorary Secretary.

Oto-Rhino-Laryngological.—Dr. Walter Crosse, Dr. A. K. Green, Honorary Secretary.

Pædiatrics.—Dr. P. A. Earnshaw, Chairman; Dr. David Jackson, Honorary Secretary.

Radiology.—Dr. Val. McDowall, President; Dr. J. R. Adam, Honorary Secretary.

Surgical.—Dr. Alan E. Lee, Chairman; Dr. R. S. Cohen, Honorary Secretary.

General Practitioner Group.

Personnel of Committee.—Dr. L. P. Winterbotham, Chairman; Dr. Garth May, Honorary Secretary-Treasurer; Dr. B. N. Adsett, Dr. R. S. Bennett, Dr. R. S. Cohen, Dr. A. D. A. Mayes, Dr. C. T. Underwood, Dr. E. Lorimer Walker.

The General Practitioner Group has been very active and was of great assistance to the Council during the year. The Council is particularly grateful for the help given in distributing, to metropolitan members, publicity literature concerning the case of the profession against the Commonwealth Government's proposals for a national medical service.

Conveners of Areas.—The following conveners were nominated to assist the committee and represent areas: Dr. H. Stanley Waters (Ashgrove), Dr. A. D. Isles (Wooloowin), Dr. H. Winterbotham (East Brisbane), Dr. C. R. Black (Coorparoo), Dr. A. J. Nicholson (Annerley), Dr. C. N.

Sinnamon and Dr. H. W. A. Forbes (Toowong), Dr. H. W. Anderson (Clayfield), Dr. P. G. D. Prentice (Sandgate-Redcliffe).

A National Health Service.

With the object of improving conditions of medical service, a positive health policy was drawn up by the Council and subsequently adopted and a copy submitted to the Federal Council for consideration.

A booklet has recently been issued enunciating the Federal Council's health policy for the nation which provides for the treatment of all classes of the people and of all phases of disease, and outlines measures for the prevention of disease by ensuring healthy conditions of living for all people.

For the middle income group the use of voluntary prepayment systems is advocated similar to that of the Medical Benefits Fund of New South Wales. It will be of interest to members of the Branch to know that negotiations are now taking place to have the New South Wales Medical Benefits Fund extended to Queensland. For details of this fund attention is drawn to Appendix 7 of the Federal Council's booklet, "A National Health Service".

It is hoped that members will do their utmost to bring before their patients and other people the policy of the medical profession in Australia to provide a medical service which will benefit the whole of the community and also be acceptable to the profession.

Members of all the Branches have taken part in a radio publicity campaign to direct the attention of the public to various aspects of the Government's plan which are considered detrimental to the liberty and interests of both patient and doctor.

A special general meeting of the Branch was held on Tuesday, March 22, 1949, which was attended by 422 members. The business of the meeting was consideration of the Government's proposals for a national health service and amendments to *The Pharmaceutical Benefits Act*.

QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION
(INCORPORATED.)

Balance Sheet as at November 15, 1949.

LIABILITIES.	£ s. d.	£ s. d.
Fixed — Loan from Queensland Medical Land Investment Company, Limited		4,650 0 0
Current—Subscriptions for Remittance to—		
British Medical Association, London	38 8 6	
Australasian Medical Publishing Company, Limited, Sydney	45 12 6	
		84 1 0
Association Funds—		
Sinking Fund	445 7 10	
Superannuation Fund	536 13 5	
	982 1 3	
Accumulation Fund	9,603 18 7	
Reserve for Entertainments	4 1 1	
		10,590 0 11
		£15,324 1 11

ASSETS.	£ s. d.	£ s. d.
Fixed, at Cost, less Depreciation—		
Land and Buildings—		
British Medical Association House — £2,256 5 11		
Bartley Street Property — 1,658 17 10		
	3,915 3 9	
Architects' Fees — preparing plans for prospective new building, at cost	300 0 0	
Library	150 0 0	
Typewriters, Bookcases, Balopticon and Furniture	95 18 0	
Bancroft Medals and Collar	4 10 0	
		4,465 11 9
Queensland Medical Land Investment Company, Limited—5950 shares of £1 each paid to 10s. each, at cost	2,975 0 0	
British Medical Agency of Queensland Proprietary, Limited — 258 shares of £1 each, fully paid, at cost	258 0 0	
		3,233 0 0
Australasian Medical Publishing Company, Limited, Sydney—		
5% Debentures, at cost	55 0 0	
3¾% Series "E" Debentures, at cost	800 0 0	
Advance not yet converted to Series "E" Debentures	78 0 9	
		933 0 9
		8,631 12 6
Current—		
Australian Consolidated Inscribed Stock, at cost—		
3¼%, maturing 1959 £1,500 0 0		
3¼%, maturing 1960 300 0 0		
	1,800 0 0	
English, Scottish and Australian Bank, Limited	3,519 15 11	
Sundry Debtors	364 0 0	
Electric Light Deposit	6 0 0	
Cash	20 12 3	
		5,710 8 2
Fund Investments—		
Sinking Fund—		
Australian Consolidated Inscribed Stock—		
£280 3¼%, maturing 1951, at cost	278 3 3	
£90 3¼%, maturing 1960,	90 0 0	
Commonwealth Savings Bank, Brisbane	77 4 7	
	445 7 10	
Superannuation Fund — Commonwealth Savings Bank, Brisbane	536 13 5	
		982 1 3
		£15,324 1 11

We have compared the above Balance Sheet with the books, accounts and vouchers of the Queensland Branch of the British Medical Association (Incorporated), and have obtained all the information and explanations we have required. In our opinion the Balance Sheet is properly drawn up to exhibit a true and correct view of the state of the Association's affairs as at 15th November, 1949, according to the best of our information and the explanations given to us, and as shown by the books of the Association. The Register of Members and other records which the Company is required to keep by the Companies Acts of 1941-1942, or by its Articles, have, in our opinion, been properly kept.

R. G. GROOM & Co.,
Chartered Accountants (Aust.),
Auditors.

Brisbane,
November 22, 1949.

H. W. HORN,
Honorary Treasurer.

QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION.
(INCORPORATED.)

Revenue Account for Twelve Months ended November 15, 1949.

EXPENDITURE.	£ s. d.	£ s. d.
November 15, 1949—		
To Branch Expenses	1,487 3 2	
„ Library Expenditure	176 19 4	
„ Depreciation of Office Equipment	21 15 3	
		1,685 17 9
„ Provision for Staff Superannuation		60 0 0
„ Federal Council — Contribution to Organization Expenses		783 6 0
„ Repatriation Medical Officers' Association — Contribution to Arbitration Appeal Expenses		50 0 0
„ British Medical Association House and Bartley Street Property Expenses	576 9 6	
Less Rents Received	567 18 0	
		8 11 6
„ Net Surplus for Year—Transferred to Accumulation Account		2,040 6 1
		£4,628 1 4

REVENUE.	£ s. d.	£ s. d.
November 15, 1949—		
By Branch and Organization Fund— Subscriptions		4,248 0 6
„ Portion of Journal Subscriptions, to be invested in Australasian Medical Publishing Company, Limited, Sydney, Series 'E' Debentures		289 2 6
		4,537 3 0
„ Interest— Commonwealth Government Inscribed Stock	53 10 0	
Australasian Medical Publishing Company, Limited— Debentures	27 11 8	
		86 1 8
„ General—Sale of Diet Sheets and Lists of Members		4 16 8
		£4,628 1 4

Dr. J. G. Hunter, General Secretary of the Federal Council, addressed the meeting and representatives of affiliated local associations and members of practically all country districts were present and expressed their views. The meeting passed a resolution of complete confidence in the Federal Council and full endorsement of its action and policy.

The meeting also authorized the establishment of the Federal Independence Fund to assist in fighting the case of the profession against proposed government regimentation. These resolutions have been put into effect in a practical manner. Members of the Branch have been very loyal to their pledge and £6054 13s. 8d. has been received from 502 members (out of a total of 802). This constitutes about £12 *per capita* of those who have subscribed. The Council is very appreciative of their support which has added to the consolidation of the profession in Australia.

The Pharmaceutical Benefits Act, 1947.

As a result of the judgement of the High Court of Australia, the amendment to *The Pharmaceutical Benefits Act*, 1947 (Section 7A), has been declared invalid in that it constitutes a violation of the liberty of the individual.

Workers' Compensation Act.

Medical Fees.—As in the past years, our agreement with the Insurance Commissioner for the payment of fees of injured workers has operated, in the main, satisfactorily.

It is a source of regret that the Commissioner has decided against continuation of payment for those who do not cease work as a result of their injury (with the exception of foreign body in the eye), but legal opinion forthcoming in the process of preparing a case to have this matter determined, puts it beyond all doubt that such a decision is within the Commissioner's competence.

Medical Fees Tribunal.

Personnel.—Dr. J. G. Wagner (chairman), Dr. Alan E. Lee (honorary secretary), Dr. R. G. Quinn, Dr. G. W. Macartney, Dr. H. S. McLelland, Dr. Norman Sherwood.

This committee met on five occasions to deal with matters concerning workers' compensation insurance medical fees and the finding in each case was that the fee was in excess of the schedule.

Medical Fees.

Life Insurance Examination Fees.—After considerable negotiation an agreement was reached between the Federal Council and the Life Offices' Association of Australasia, that the fee for examination of candidates requiring the use of the extended form shall be £1 11s. 6d. as from August 1, 1949.

The Federal Council had sought an increase from £1 1s. to £2 2s., but had accepted the compromise in view of £1 11s. 6d. being the rate payable in other parts of the British Commonwealth.

The State Government Insurance Office has also increased its fee accordingly.

University of Queensland.

British Medical Association (Queensland Branch): Queensland Medical Students' Loan Fund.—The personnel of the committee of administration is as follows: Dr. C. A. Thelander (chairman), the Dean of the Faculty of Medicine (Dr. E. S. Meyers), Professor H. J. Wilkinson, Professor Alex. Murphy, Dr. Arnold W. Robertson and a representative of the University of Queensland Medical Society (nominated annually by the society), and the ex-officio members of the Council. The fund now stands at £646 1s. 1d. During the year £60 10s. 9d. was donated by members of the Branch.

Honorary Associate Members of the Branch.—The number of honorary associate members of the Branch is now 178, of whom 78 were elected during the year.

Post-Graduate Medical Education Committee.—The representatives of the Branch on this committee are Dr. Harold Love, Dr. Alan Lee and Dr. J. R. Adam. The activities of the committee have been extended during the year, and the first number of *The Queensland Post-Graduate Medical Journal* is now being printed. This journal will be distributed, free of charge, to all doctors in Queensland. New centres visited by lecturers are Charleville, South Coast and Warwick, and arrangements are being made to send lecturers to Bundaberg and Inglewood. Eminent visitors from overseas during 1949 were: Professor L. S. P. Davidson, Professor of Medicine in the University of Edinburgh; Dr. W. D. Hobson, Medical Director, McIntyre Research, Limited, Canada; Professor F. A. E. Crew, Professor of Public Health, University of Edinburgh; Professor Alexander Kennedy, Professor of Psychiatry of Durham University.

Post-graduate week was held from May 28 to June 3. The visiting lecturers were Dr. T. M. Greenaway and Mr. A. E. Coates. Other interstate lecturers during the year were Dr. E. A. North, of the Commonwealth Serum Laboratories, and Dr. A. H. Ennor, Professor of Biochemistry in the Australian National University.

Dr. P. H. Macindoe was appointed Director of Post-Graduate Studies in July, when Dr. Keith A. Moore tendered his resignation prior to his departure for England.

Harold Plant Memorial Prize.—Notification was received that it had been decided by the Senate not to award a prize for 1948 as there was no candidate of sufficient merit.

Memorial Prize of the British Medical Association (Queensland Branch).—The Memorial Prize was awarded to Clifford Robert Lulham for 1948.

William Nathaniel Robertson Medal.—Advice was received from the Registrar of the University of Queensland that this medal was won by John Joseph O'Sullivan in 1948.

Australasian Medical Congress (British Medical Association), Seventh Session, Brisbane, May 27 to June 2, 1950.

Arrangements for the seventh session are reported to be progressing satisfactorily. Professor Alex. Murphy, Professor of Medicine in the University of Queensland, has been appointed president. Dr. W. J. Saxton is the honorary general secretary of congress and Dr. David Jackson and Dr. G. A. McLean are assistant honorary secretaries. Mrs. E. Maughan was appointed secretary to the Executive Committee.

"The Medical Journal of Australia."

The Editor of THE MEDICAL JOURNAL OF AUSTRALIA, Dr. Mervyn Archdall, paid a visit to Brisbane and a hearty

QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION
(INCORPORATED.)

GENERAL FUND.

Statement of Receipts and Payments for Twelve Months ended November 15, 1949.

RECEIPTS.	£ s. d.	£ s. d.
November 16, 1948—		
To Funds at November 16, 1948—		
English, Scottish and Australian Bank, Limited—Current Account	1,767 7 7	
Cash in Hand	1 7 9	
		1,768 15 4
November 15, 1949—		
„ Subscriptions—		
Queensland Branch Subscriptions	2,906 6 0	
Organization Fund, Queensland Branch	1,341 14 6	
For Remittance to British Medical Association, London	992 2 6	
For remittance to THE MEDICAL JOURNAL OF AUSTRALIA, Sydney	880 2 6	
		6,120 5 6
„ General—		
Rents—		
British Medical Assoc. House . £297 10 0		
Bartley Street Property 218 8 0		
	515 18 0	
Interest on Commonwealth Inscribed Stock	58 10 0	
Australasian Medical Publishing Company, Limited—Interest on Debentures	27 11 8	
Sale of Typerwriters	26 10 0	
Sale of Diet Sheets and Sundries	4 16 8	
		633 6 4
		£8,522 7 2

PAYMENTS.	£ s. d.	£ s. d.
November 15, 1949—		
By Amounts remitted on account of Subscriptions collected to—		
British Medical Association, London	983 13 3	
Australasian Medical Publishing Company, Limited, Sydney, for publication of Journals	585 10 0	
Applied to Series "E" 3½% Debentures	289 2 6	
		1,858 5 9
„ Federal Council — Contribution to Expenses		783 6 0
„ Branch Expenses—		
Salaries, Audit and Honoraria	859 4 6	
Publicity *re* Pharmaceutical Benefits and National Medical Service Act	175 10 0	
Printing and Stationery	158 1 3	
Postages and Duty Stamps	142 4 3	
Bank Charges, Meeting Expenses and Sundries	68 4 0	
Expenses, Bancroft Oration	45 6 6	
Telephone	31 9 10	
Gas and Lighting	16 16 11	
		1,496 17 3
„ British Medical Association House Expenses—		
Rates to December 31, 1949	161 11 4	
Cleaning	109 4 10	
State Land Tax	10 18 9	
Repairs and Maintenance	73 10 8	
Insurance	6 0 9	
		361 6 4
„ Bartley Street Property—		
Rates to December 31, 1949	24 18 9	
Repairs and Maintenance	35 12 8	
Legal Expenses	21 15 9	
Insurance	16 4 4	
Inspection Fee	2 2 0	
		100 13 6
„ General—		
Library Expenditure	176 19 4	
Transfer to Superannuation Fund Account with Commonwealth Savings Bank	60 0 0	
Net Cost, British Medical Association Ball	11 9 0	
Purchase of Furniture and Equipment—		
Furniture £8 2 5		
Typewriter 32 4 0		
Tecnico Floor Polisher 22 8 8		
Filing Cabinet 20 6 9		
	83 1 10	
Repatriation Medical Officers' Association — Contribution to Arbitration Appeal Expenses	50 0 0	
		381 10 2
„ Funds at November 15, 1949—		
English, Scottish and Australian Bank, Limited, Brisbane	3,519 15 11	
Cash	20 12 3	
		3,540 8 2
		£8,522 7 2

welcome was extended to him at a meeting of the Council, which he attended, on March 25, 1949.

Federal Medical War Relief Fund (1939-1946 War).

Personnel of the Local Committee of Management.—Dr. J. G. Wagner (chairman), Dr. F. W. R. Lukin (honorary secretary), Dr. J. V. Duhig, Dr. Milton Geaney. In August, Dr. J. G. Avery tendered his resignation owing to his departure from the State. Dr. Love was appointed in his stead.

Substantial assistance has been rendered to Queensland beneficiaries, from whom applications were received.

The local committee would be pleased to submit applications to the trustees, on behalf of any persons who are eligible to participate in the benefits of this fund. It has been decided by the Federal Council to keep the fund open for an indefinite period and subscriptions are still acceptable.

Federal Council of the British Medical Association in Australia.

Owing to his resignation as a representative of the South Australian Branch, Sir Henry Newland's presidency of the Federal Council, an office which had been held by him for over sixteen years, automatically ceased.

The British Medical Association in Australia owes a great debt to Sir Henry Newland, as during the period of his leadership the Federal Council has gained recognition as the authoritative body to speak for the whole of the profession in this country. We congratulate Mr. Victor Hurley upon his appointment as President of the Federal Council.

Meetings of the Federal Council were held in Sydney on December 11 to 13, 1948, in Melbourne from March 1 to 4, 1949, and in Sydney on July 22 to 25, 1949. The Branch was represented at these meetings by Dr. Alan E. Lee and Dr. H. W. Horn.

We were very pleased to receive an official visit from Dr. J. G. Hunter, General Secretary of the Federal Council, who came to Brisbane to be present at the special general meeting of the Branch held on March 22, 1949.

British Medical Agency of Queensland Proprietary, Limited.

The agency has experienced another busy year and continues with the endeavour to render service to members.

Additional activities have involved an increase in staff within the limited accommodation available and further expansion is unlikely to become practicable until new premises are constructed.

The estates of deceased members of the Branch have been assisted by the services of the agency in respect to the winding up of professional affairs.

The financial results of the year are reported to be satisfactory.

Queensland Medical Finance Proprietary, Limited.

Inquiries for financial assistance have been limited; the large majority of members taking up partnerships or entering into private practice have relied on their own resources.

The company is ready and prepared to consider applications to assist members in the purchase of medical practices.

Social.

As usual, during post-graduate week the annual ball was held in Lennon's Hotel ballroom on Wednesday, June 1, 1949. The official guests included Sir Henry Newland, Dr. and Mrs. T. M. Greenaway, and Mr. and Mrs. A. E. Coates. It was a very friendly and enjoyable function, if somewhat crowded.

Prior to the Joseph Bancroft Oration, on Friday, June 3, the Council entertained at dinner at the National Hotel, the Orator, Sir Henry Newland; Dr. T. M. Greenaway; Mr. A. E. Coates; the chairman of the State Committee of the Royal Australasian College of Physicians, Dr. L. J. Jarvis Nye; the chairman of the State Committee of the Royal Australasian College of Surgeons, Dr. Alan E. Lee; the chairman of the University of Queensland Post-Graduate Medical Education Committee, Dr. A. V. Meehan; and the director, Dr. Keith A. Moore.

Conclusion.

A very large portion of this year's work has been of a medico-political nature and we owe a debt of thanks to our two federal delegates, Dr. Horn and Dr. Lee, for the great amount of work they have done for the association and for the time they have given up to attend the Federal Council meetings.

One of the highlights of the year was the extraordinary general meeting held in March, when more than two-thirds of the profession attended. Members from all parts of the State were present and many spoke at this meeting.

For the first time in Queensland British Medical Association history the Press were invited to the meeting. I would like to place on record my deep appreciation of the cordial relations that have existed between the Press and the British Medical Association and for the publicity which they have given to our fight against socialization of medicine.

The relations between the British Medical Association and the State Government have been most cordial, and I feel that as a result of our several conferences with the Department of Health and Home Affairs both sides achieved something to the mutual benefit of each party.

To the profession as a whole, I desire to thank you for the honour which you conferred upon me by electing me President of the Queensland Branch of the British Medical Association, and I also wish to express my appreciation of the assistance rendered to me by members of the Council during that period. It is not always possible for one individual to keep track of all of the activities of the association, and during my term I was most fortunate to have on the Council a number of members who could supply information and advice on some knotty problems.

The Council and the profession as a whole owe a debt of gratitude to Mrs. Spooner and her staff for her untiring work and the extremely long hours which she is called upon to work, particularly with Council meetings which often last until very late at night.

To my successor, Dr. Love, I extend my best wishes and I feel sure that he will have the same loyal cooperation which I was fortunate enough to have during my term.

B. L. W. CLARKE,
President.

BALANCE SHEET AND FINANCIAL STATEMENT.

The balance sheet and financial statement as at November 15, 1949, which had previously been circulated among members, was adopted on the motion of Dr. H. W. Horn, seconded by Dr. Noel Gutteridge. The statement is published herewith.

MEMORIAL ROLL.

With the members standing, the President read the names on the Memorial Roll of the Branch.

ETHICS COMMITTEE.

Dr. Val McDowall, Dr. L. J. J. Nye, Dr. J. J. Power, Dr. M. G. Sutton, Dr. R. G. Quinn, Dr. N. Sherwood and Dr. G. W. Macartney were elected members of the Ethics Committee.

ELECTION OF OFFICE-BEARERS.

The President announced the results of the election of office-bearers and members of the Council.

President: Dr. Harold Love.
President-Elect: Dr. Arnold Robertson.
Past President: Dr. B. L. W. Clarke.
Councillors: Dr. B. N. Adsett, Dr. Felix Arden, Dr. Ronald S. Bennett, Dr. K. B. Fraser, Dr. G. V. Hickey, Dr. Harold Horn, Dr. J. R. S. Lahz, Dr. A. E. Lee, Dr. F. W. R. Lukin, Dr. Alex Mayes, Dr. H. S. Patterson, Dr. W. H. Steel, Dr. Lorimer Walker, Dr. J. G. Wagner.

ELECTION OF AUDITORS.

Messrs. R. G. Groom and Company were reelected auditors for the ensuing year.

INDUCTION OF PRESIDENT AND PRESIDENT'S ADDRESS.

Dr. B. L. W. Clarke vacated the chair in favour of Dr. Harold Love, the incoming President. Dr. Love paid a tribute to Dr. Clarke on his competent handling of the affairs of the Branch during a difficult year, and thanked members for his election. Dr. Love then delivered his president's address (see page 173).

VOTES OF THANKS.

Votes of thanks to the retiring councillors and to Mrs. Spooner and the secretarial staff were carried by acclamation.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on November 10, 1949, at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, DR. J. KEMPSON MADDOX, the President, in the chair.

Psychological Symptoms Associated with Dysmenorrhœa and the Climacteric.

DR. A. A. MOON read a paper entitled "Dysmenorrhœa and the Climacteric" (see page 174).

DR. A. T. EDWARDS read a paper entitled "Psychological Implications of Dysmenorrhœa and the Menopause" (see page 178).

MR. LATHAM asked Dr. Edwards whether he was called upon to treat many women suffering from dysmenorrhœa.

DR. D. W. H. ARNOTT, in reply to Mr. Latham, said that he had never been called upon specifically to treat a patient suffering from dysmenorrhœa, although it was a frequent complaint in nervous women. He described both papers as interesting, and said that they dealt well with a difficult and nebulous subject. In connexion with the menopause, mental deviations were common and were frequently treated with ovarian hormone, but he thought that it gave but little relief for these symptoms. The menopause frequently produced mental morbidity in women, but it was not clear exactly how this was brought about. At the menopause the scales fell from the eyes of a woman, and she saw her life's situation clearly, generally for the first time. If she was satisfactorily placed in life, in that she had a loving husband and children and grandchildren who were dependent on her, her future was promising. To many women, however, at this time, life was fruitless and empty, and if one added to this an indifferent comrade and an inept lover as a husband, was it any wonder that woman's soul revolted with bitterness and hostility or often found refuge in mental ill health? Her future welfare depended on her ability to change her attitudes and her life in general.

DR. D. R. MORGAN asked whether dysmenorrhœa was more common in single than in married women. Dr. Moon had

referred to the symptoms of a healthy country girl. Dr. Morgan said that it was commonly believed that young women who worked in factories or sat all day in an office were more inclined to spasmodic dysmenorrhœa than those who lived open-air lives. He also asked whether dysmenorrhœa was more uncommon among the less civilized peoples. Finally he asked Dr. Edwards whether he regarded involutional melancholia as a distinct entity.

DR. J. C. LOXTON referred to the two groups of dysmenorrhœa, primary and secondary, and said that he would deal only with the former. Of the cases of primary dysmenorrhœa which were encountered in practice, some 75% to 80% had a marked psychological basis. This had been proved during the recent war. Many women had gone into camp in the different service units, and symptoms from which they might have suffered disappeared when they carried on with their jobs. The remaining 20% of women whose dysmenorrhœa had little or no psychological basis were examined, and some endocrine therapy was ordered for them. Perhaps œstrin would produce a satisfactory result. Sometimes symptoms recurred, and dilatation of the *os uteri* would be carried out, or even curettage of the uterus by some. Of those submitted to such procedures, perhaps 50% would be cured. In half of these cases the cure would be permanent, and in the remainder recurrence usually took place. The problem was one of some difficulty, but Dr. Loxton thought that as knowledge of the progress of civilization increased, medical practitioners would be able to deal with the sufferers in a more satisfactory way.

DR. S. BENEDEK said that he joined Dr. Morgan in expressing appreciation to both speakers for their interesting and stimulating papers, and asked Dr. Edwards what was the incidence of dysmenorrhœa in mental hospitals, as, if the condition was an organ neurosis, its occurrence must be very rare.

DR. J. KEMPSON MADDOX said that he looked at the question of dysmenorrhœa from the point of view of a physician. The condition was an organ neurosis, a painful dysfunction of a hollow organ. Such neuroses were common with other hollow organs associated with the alimentary and renal tracts. He thought that possibly the condition might be elucidated on comparative physiological lines. Dr. Moon had welcomed the assistance of the psychiatrist, and had said that in certain circumstances a psychiatrist should always be consulted. That sort of collaboration had not existed a few years earlier. Dr. Maddox referred to the woman with primary dysmenorrhœa of the type described by Dr. Edwards, and said that she generally complained of other anxiety symptoms and had more than the average share of femininity. It had been said that Negro women did not suffer from dysmenorrhœa, but that American Negresses suffered as acutely as American white women. Dr. Maddox thought that the use of the word "change"—"change of life"—had dire psychological consequences. It was only by a process of public education that the use of such terms would disappear. The really fascinating part of the subject was why the menopause should arise. Was it initiated by a time-limited endocrine imbalance, or by some phasic irregularity in the control of the nervous system? Equally fascinating, but obscure, were the associated changes that sometimes occurred—obesity and hyperglycæmia. Some quantitative disturbance of the action of the pituitary hormones was supposed to take place, but it was not clear whether that gland alone was capable of producing such diverse and far-reaching changes.

Dr. Edwards, in reply, said that he did not see many patients suffering from dysmenorrhœa. He had never been called upon to treat a patient for that condition alone. Sometimes dysmenorrhœa occurred with dyspareunia. If the dyspareunia was treated and it cleared up, the dysmenorrhœa disappeared with it. He agreed with Dr. Arnott that the results of the treatment of menopausal symptoms with œstrogens were disappointing, but that did not mean that there was no place for œstrogens in the treatment of that condition—psychiatrists saw only the failures of hormonal treatment given by physicians. Dr. Morgan had asked whether there was a difference between the menopausal symptoms of married and unmarried women. He thought that the symptoms occurred in equal proportions in both groups. The manifestations of the menopause might be different, and the complaints might be different in the two groups. Moreover, the guilt reactions were different. He thought that involutional melancholia was a distinct entity. It had a different symptomatology, different prepsychotic personality and different outcome. In reply to Dr. Benedek, he said that he had been attached to the Mental Hospitals Department for twenty-two years, and he had never seen a patient in bed because of dysmenorrhœa. There was no reason why they should suffer from symptoms of psychoneurotic origin, because they had already achieved a flight from reality.

Dr. Moon referred to dysmenorrhœa as evidenced by sedentary and rural workers. He thought that the condition was one affecting all classes of the community. At the same time he had been impressed with the number of healthy country girls who suffered. He referred to what had been said about civilized and uncivilized communities, and said that he did not know whether the supposed prevalence of menopausal symptoms in civilized as opposed to uncivilized communities was greater.

Dr. Maddox, from the chair, thanked the speakers for their papers, and called for a vote of thanks, which was carried by acclamation.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

General Revision Course.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that its annual general revision course will be held during the two weeks preceding Easter, beginning March 27, 1950. The course is under the supervision of Dr. R. L. Harris and is designed to give a general review of all aspects of medicine and surgery.

As seminars, at which short papers are presented by a number of lecturers dealing with various aspects of the subject, have proved very popular in previous courses, they have been made a feature of this year's programme and will include seminars on gynæcology, toxæmias of pregnancy, acute abdominal conditions, poliomyelitis, tuberculosis and obesity, and a panel discussion on hypertension. It is also hoped to arrange a demonstration on television.

Provision has also been made so that the wives of those attending the course will have an opportunity of meeting others attending, and a number of social functions have been arranged.

Fees for attendance are as follows: full course, £5 5s.; mornings or afternoons only, £3 3s.; one week only, £3 3s. Early application, enclosing remittance, should be made to the Course Secretary, the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 5238, BW 7483. Telegraphic and cable address: "Postgrad, Sydney."

THE ROYAL MELBOURNE HOSPITAL.

Lectures in Anæsthesia.

DR. RONALD JARMAN, of London, will give the following two lectures in the main lecture theatre (ground floor), The Royal Melbourne Hospital, at 8 p.m. All interested in anæsthesia are invited to attend.

Wednesday, February 22, 1950: "Nearly Twenty Years' Experience of Intravenous Anæsthesia."

Friday, February 24, 1950: "General Survey of Anæsthesia in Britain Today."

Correspondence.

PULMONARY TUBERCULOSIS IN THE MENTAL HOSPITALS OF WESTERN AUSTRALIA.

SIR: In their article on pulmonary tuberculosis in the mental hospitals of Western Australia in today's journal, Dr. Frank Prendergast and Dr. Alan King conclude that there is need to survey the patients in all mental hospitals in order to discover sufferers from tuberculosis. They base their conclusion on an X-ray survey conducted by themselves and upon certain investigations in other countries, the earliest of which was reported in 1941.

In a communication published in THE MEDICAL JOURNAL OF AUSTRALIA on June 1, 1940, I showed that the incidence of tuberculosis infection in a mental hospital near Sydney was

very much higher, especially amongst younger inmates, than in the population at large. This information was based on a tuberculin survey then recently completed, and I made the suggestion that the intracutaneous tuberculin test would be found to be a valuable contributory method in the economical solution of the public health problems occasioned by tuberculous disease in mental hospitals.

In an article on the control of tuberculosis published in THE MEDICAL JOURNAL OF AUSTRALIA on September 23, 1944, I mentioned that several nurses and other workers in mental hospitals in New South Wales, whom I had seen professionally, had never been tested with tuberculin or X-rayed, notwithstanding evidence suggesting that the proportion of mental hospital patients suffering from tuberculosis was much greater than in the general hospital population. The oversight in respect of the mental hospital nurses has now been remedied, I understand, but I am not aware of any investigation to discover and treat sufferers from tuberculosis in New South Wales mental hospitals.

I hope that the survey of Dr. Prendergast and Dr. King may bear fruit sooner than mine has done.

Yours, etc.,

185 Macquarie Street, Sydney, January 21, 1950. DOUGLAS ANDERSON.

"THE CITY OF DREADFUL NIGHT."

SIR: It was recently my sad privilege to sit at night with a dying friend. The shortage of private hospital accommodation for the chronically ill in Brisbane and the scarcity of trained nurses had thrown such a burden on my friend's wife that some of his friends had taken turns to help her obtain some much-needed rest.

During the first night the patient was dozing—his pain controlled to some extent by analgesics, till suddenly at about 2.30 a.m. the cocks began to crow—the din commencing first about 100 yards away. Soon so many were crowing that it was impossible to count the number. The dying man stirred and muttered: "'The city of dreadful night', when your turn comes, go to Melbourne to die in peace." The unholy row continued for an hour and a half; there was then a period of quiet, during which the patient dozed till about 4.30 a.m., when again the cacophony started. Again the dying man was roused. I offered morphine, but as the patient was feeling no pain, this was refused. He said: "Give it to the roosters and I will then sleep."

My friend's wife said that this was a nightly occurrence, and her husband had little sleep after 2.30 a.m. each day.

I visited the neighbours who owned the nearest roosters. They were apparently sympathetic, but obviously, being themselves in rude health, didn't believe me when I told of the additional misery their fowls were causing to one already bearing so much. However, they must have shut their roosters up for the next night. After that they forgot.

My friend died while we watched, in the middle of the usual din from the roosters. Since then, with my friend's misery and his wife's anguish fresh in my mind, I have talked to people who keep backyard poultry. I know a little about fowls, having lived on a farm in my youth. These cockerels are not kept for breeding purposes but for eating. Few suburban hen-keepers breed their own stock, buying day-old chicks to replenish their stock of layers. The backyard rooster apparently has a low fertility rate. The belief is also held that cocks are better to eat than hens. I had thought that everyone knew that a hen is preferable to a caponized cock, and cow-beef better than beef from a bull. However, suburbia thinks the bull is castrated solely to tame him.

When I asked the backyard poultry-keepers why they didn't raise pullets for eating I was told that this was wasteful. But whether this referred to eggs or to the fact that hen chickens are a penny each or so dearer than male chickens I know not. Surely the difference of a shilling or so when a dozen chickens are bought to rear for the table should not be the cause of sleepless nights for the ill, the aged and the dying.

My late friend's house is situated only two miles from the General Post Office, and only about one in three households keeps fowls. What it must be like in the more distant suburbs where every house has its chanticleer—or two or three—I hate to imagine.

The additional misery of broken sleep and the realization of the pain that is inflicted on sufferers is not due to conscious cruelty, but to lack of imagination and the indifference of some people to the pains and unhappiness of others. This fact is obvious when one reads letters in the local Press recently in which the "pro-roosters" accused the insomniacs of being neurotic.

To deal with such an uncivilized situation requires propaganda. But by whom should a campaign of such propaganda be initiated? Would it be too much to ask if the Red Cross Society would add one more humanitarian service to the many that they have already in hand, and try to help those unfortunate enough to be ill or to die in Brisbane?

Yours, etc.,

Brisbane, January 5, 1950. "GALLUS."

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

Plenary Session.

THE subject to be discussed at the plenary session of the Australasian Medical Congress (British Medical Association), Seventh Session, Brisbane, 1950, is "Rheumatism and its Sequelæ". The plenary session will be held on the morning of Tuesday, May 30, and will inaugurate the scientific proceedings of congress.

It has been decided that the plenary session will take the form of a "panel discussion". In such a discussion a group of selected authorities sit at a table on the platform under the direction of a chairman. The chairman reads out questions submitted previously by such members of congress as are to do so, and selects one or more of the panel to answer each question. The questions are arranged in order beforehand, so that a coordinated over-all picture of the subject under discussion is obtained; but instead of the long formal papers, the information is provided by means of succinct comments by a number of experts speaking for short periods. It is thought that such an arrangement will give a much more lively and interesting review of the important subject under discussion, as it will exclude unnecessary padding and enable the subject to be dealt with very widely with the expression of many views.

All members of congress are invited to submit their questions on rheumatism and its sequelæ. These questions should be sent as soon as possible direct to the Honorary General Secretary, Australasian Medical Congress (British Medical Association), British Medical Association House, Wickham Terrace, Brisbane.

Obituary.

LUCY EDITH GULLETT.

WE are indebted to Dr. Kathleen Cuningham for the following appreciation of the late Dr. Lucy Edith Gullett.

Lucy Edith Gullett was one of the most outstanding of the pioneer women of the medical profession. Born in 1876 of literary parents, she had a compelling personality, wide literary tastes, a polished command of the English language and a wonderful sense of humour. She was kind and sympathetic, especially to those less fortunate than she, and, although gifted with a pretty turn of wit, she did not use this at the expense of any person. Lucy Gullett graduated in medicine in 1902, later serving a term of residency at the Brisbane Children's Hospital. From 1906 to 1911 she practised in the Bathurst district and then returned to Sydney to practise in Kirribilli. During the first World War she served in a French hospital at Lyons, returning to Australia in 1918 after the end of the war. She was appointed honorary physician to the Renwick Hospital for Infants and honorary medical officer to the baby health centres, and was an active member of the District Nursing Association all her life.

In 1921 Dr. Gullett went to Melbourne for the jubilee celebrations of the Queen Victoria Memorial Hospital, which is staffed entirely by women. So impressed was she by the work done in this hospital and the benefits derived from it by women doctors practising in Victoria, that she came home determined to see established a similar hospital in Sydney. She interested her friends and called a meeting of her fellow practitioners, amongst whom were Dame Constance D'Arcy, Dr. Harriett Biffin, Dr. Mary Burfitt, Dr.

Margaret Harper, Dr. Susie O'Reilly and Dr. Emma Buckley. These women and their friends subscribed £1000 towards the establishment of a hospital for women and children, to be staffed by women. A house was purchased in Landsdowne Street, Surry Hills, and opened in 1922 as an out-patient department, being known as the New Hospital for Women and Children. Later the name was changed to The Rachel Forster Hospital for Women and Children. In 1926 a move was made to larger premises, in 1933 these were added to, and in 1939 it was decided to tender for plans for the present site of the 120-bed hospital now standing in Pitt Street, Redfern.

Through all these years, Lucy Gullett was indefatigable in her work for the hospital. She was an active physician on the staff and, in addition, was vice-president of the Board of Directors, and chairman of the honorary medical staff until 1942. She organized many of the centres established in the various suburbs and attended their annual meetings, encouraging their excellent work by her dynamic personality and wonderful sense of humour. Her inspiration

and enthusiasm always resulted in an increase in the output of the centre after one of her visits to it. She was always thinking of the hard lot of the working man's wife, and the busy woman who ran her home and did an outside job as well—she insisted on opening an out-patients' session on one night a week, especially for working women and for those with young families who could not spare time to attend to their own health. She herself staffed this for many years, and when some of her colleagues suggested doing away with the Tuesday night session, she fought strenuously to have it continued.

In 1939, plans were drawn up for the present hospital; tenders were called for, and in June, 1940, a suitable tender was put to the Board of the hospital for acceptance. A special meeting was held at noon one day to consider whether this tender should be accepted or whether, in view of the grave world situation, the building of the hospital should be deferred until we knew what was going to happen to the British Empire. The chairman came to the meeting armed with a mid-day edition of the evening paper, and across it was printed in letters three inches high: "Last of the Channel Ports Falls." This cast a gloom upon the meeting and members of the Board were fearful of taking the step, which would involve them in heavy expenditure. Lucy Gullett was undaunted by the world crisis and eventually spoke up in these words: "Well, I think we should go on with the building of the new hospital. If the British Empire falls, it won't matter on what we have spent our money; but if it doesn't, we will have our hospital." This statement swayed the meeting; the tender was accepted and the building was erected.

In December, 1941, Lucy Gullett's dreams were realized, and the present hospital building was opened by the Lady Wakehurst. In her speech on the opening day, Dr. Gullett said that it was one of the greatest moments of her life. Many of her friends had told her she should be well satisfied with her efforts. But she said that she was not satisfied—having seen the hospital open, she was going to work to establish a convalescent home to be run in conjunction with the main hospital.

In 1942, after twenty years as chairman of the staff, Dr. Gullett wished to retire from the position. The honorary medical staff gave her a cheque for £125 to form the nucleus of a fund for her convalescent home. Little did we dream at that time that in less than five years the home would be open, ready to receive patients, and bearing her own name, "The Lucy Gullett Convalescent Home".

Those of us who worked with her through the years of the establishment of the hospital will miss her, the thousands who have benefited directly or indirectly through her work will mourn her, but these two living memorials, the Rachel Forster Hospital and the Lucy Gullett Convalescent Home, will keep alive for all time the memory of a truly great woman.

Dr. Guy Griffiths writes: Lucy Gullett was the daughter of a distinguished father, the Honourable Henry Gullett, M.L.C., editor of *The Sydney Morning Herald* and of *The Daily Telegraph*, philanthropist and Shakespearean scholar. From him she inherited a love of literature, especially of English poetry and of the authorized version of the Hebrew psalms. Her father had intended to present a Shakespearean memorial to Sydney, and after his death Dr. Lucy and her sisters commissioned Bertram Mackennal to produce the beautiful and stately statuary group which now adorns the entrance to the Domain in front of the Public Library.

She was resolute and patriotic; she served as a surgeon in France in the 1914-1918 war. She was capable both as a surgeon (she won the prize for clinical surgery when she graduated M.B. in December, 1900) and as an administrator.

Above all she was kindly: her goodness of heart won her the lasting devotion of many women friends and patients. It was indeed touching to see their affectionate care and concern during her last sad illness.

Mentally she was self-reliant: she would give courteous attention to the opinions of others, but no slavish subservience to dogma by whomever propounded.

JAMES SYDNEY WOOLNOUGH.

WE regret to announce the death of Dr. James Sydney Woolnough, which occurred on February 1, 1950, at Hazelbrook, New South Wales.

The Royal Australasian College of Physicians.

COURSE IN ADVANCED MEDICINE.

THE Victorian State Committee of The Royal Australasian College of Physicians has arranged a six weeks' course in advanced medicine to be conducted at Prince Henry's Hospital, Melbourne, on Tuesday, Thursday and Friday afternoons of each week from March 28 to May 12, 1950. The course is suitable for those preparing for examinations for higher degrees in medicine or for physicians in general practice desiring a refresher course.

On Tuesdays and Thursdays it is hoped to have patients available for examination from 1.30 to 2.15 p.m. A round of these patients will follow from 2.15 to 3.30 p.m. From 4 to 4.15 p.m. there will be a lecture on a listed subject, and from 5 to 5.30 p.m. a demonstration on a clinical, pathological or radiological subject. On Fridays ward rounds only will be held, commencing at 2.30 p.m. A detailed syllabus is available.

The course will be limited to sixteen students, and a fee of £12 12s. will be charged. Applications should be lodged by March 7, 1950, with Dr. Eric Clarke, c.o. the Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1, from whom further inquiries may be made.

Australian Medical Board Proceedings.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts, 1939 to 1948*, of Queensland, as duly qualified medical practitioners:

Benn, Keith McLeod, M.B., B.S., 1949 (Univ. Melbourne), c.o. Hospitals Board, Cairns.

Meagher, Kevin James, M.B., B.S., 1941 (Univ. Melbourne), Brisbane Mental Hospital, Goodna.

Thompson, Leslie Joseph, M.B., Ch.B., 1912 (Univ. New Zealand), F.R.C.S. (Edinburgh), 1924, c.o. Mr. R. G. Thompson, Bruce Furse and Associates, 17 O'Connell Street, Sydney, New South Wales.

Ashworth, Alan, M.B., Ch.B., 1925 (Univ. Liverpool), M.R.C.S., L.R.C.P. (London), 1926, M.D., 1939, D.T.M., 1926, D.P.H., 1932 (Univ. Liverpool), City Hall, Brisbane.

Forsayeth, Richard Martin, M.R.C.S. (England), L.R.C.P. (London), 1926, Atherton.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales as duly qualified medical practitioners:

Blackwood, Forsten James Allan Helge, M.B., B.S. (Univ. London), M.R.C.S. (England), L.R.C.P. (London), 1945, F.R.C.S. (England), 1947, Royal North Shore Hospital, St. Leonards.

Geates, John Binning, M.B., Ch.B., 1943 (Univ. Glasgow), 8 Oberon Crescent, Gordon.

Harrison, William Rhodes, M.R.C.S. (England), L.R.C.P. (London), 1944, c.o. Dr. Browne, Mittagong.

Nagle, Francis Charles, M.B., B.S., 1949 (Univ. Melbourne), 5 Mistral Avenue, Mosman.

Taylor, James Clarke, M.B., Ch.M., 1934 (Univ. Glasgow), Headquarters, Eastern Area, Royal Australian Air Force, Bradfield Park.

Watson, Joseph Lee, M.B., Ch.B., 1943 (Univ. Edinburgh), D.P.H., 1949 (Univ. Edinburgh), H.M.A.S. *Kuttabul*, Garden Island, Sydney.

The following additional qualifications have been registered:

Anderson, Neville Arthur, c.o. London Missionary Society, Port Romilly, Papua (M.B., B.S., 1945, Univ. Sydney), D.T.M. and H., 1948 (Univ. Sydney).

Basil-Jones, Brian James, Royal Hospital for Women, Paddington (M.B., B.S., 1939, Univ. Sydney), D.C.P., 1949 (Univ. Sydney).

McDonnell, Laurence Edward, 13 Gilbert Park, Manly (M.B., B.S., 1944, Univ. Sydney), D.A. (R.C.P. and S., England), 1949.

Rogers, Peter Augustine, 235 Macquarie Street, Sydney (M.B., B.S., 1946, Univ. Sydney), Dip. Ophth., 1949 (Univ. Sydney).

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Cramer, Erle William, M.B., B.S., 1948 (Univ. Sydney), 47 Shirley Road, Wollstonecraft.

Donald, Gordon Frederick, M.B., B.S., 1948 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Reimer, John Edward, provisional registration, 1949 (Univ. Sydney), "Rosemont", 25 Shirley Road, Roseville.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JANUARY 21, 1950.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	7(6)	1	..	..	..	..	..	8
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	1	1	2	..	..	4(1)	..	..	8
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	1(1)	..	..	..	..	..	1
Diphtheria	3	4(4)	7(1)	1(1)	..	..	..	..	15
Dysentery (Amœbic)	*	1(1)	..	..	..	..	..	..	1
Dysentery (Bacillary)	*	..	11(11)	..	..	..	..	..	11
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	..	..	*	..	..	..
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	..	*	*	..	*	*	..	..
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	..	*	*	*	*	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*b*)	*	1(1)	1(1)	..	2	..	..	..	4
Measles	*	*	*	9	*	*	..	..	9
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	22(15)	8(5)	..	38(31)	1	..	..	..	69
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	..	..	..	2(1)	..	..	..	..	2
Rubella(*c*)	..	*	1	*	2(1)	1	..	..	4
Scarlet Fever	13(6)	20(9)	5(2)	2(1)	4(1)	1(1)	..	2	47
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	1(1)	*	*	*	*	*	1
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	38(25)	15(9)	8(4)	4(2)	16(8)	3(1)	..	..	84
Typhoid Fever(*e*)	1	..	..	..	..	..	..	..	1
Typhus (Endemic)(*f*)	..	..	2	..	..	..	..	..	2
Undulant Fever	..	1	..	..	..	..	..	..	1
Weil's Disease(*g*)	*	*	*	*	*	*	*	*	..
Whooping Cough	*	*	*	15(7)	*	*	..	..	15
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Northern Territory.
* Not notifiable.

(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

Grahame, Barbara Ellen, provisional registration, 1949 (Univ. Sydney), Cessnock District Hospital, Cessnock, New South Wales.

Abrahams, John Joseph, provisional registration, 1949 (Univ. Sydney), Ryde District Soldiers' Memorial Hospital, Eastwood.

Walker, Thomas Bridson, provisional registration, 1949 (Univ. Sydney), 26 Burlington Road, Homebush.

Hopkins, Peter Frederick, M.B., B.S., 1948 (Univ. Sydney), c.o. Commercial Banking Company of Sydney, Limited, Gunnedah, New South Wales.

Bern, Max Heinz, provisional registration, 1949 (Univ. Sydney), 13 Elizabeth Bay Road, Elizabeth Bay.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association:

Burdon, Kenneth Roy, M.B., B.S., 1949 (Univ. Adelaide), 10 Fisher Street, Tusmore.

Bennett, John Barkley, M.B., B.S., 1947 (Univ. Adelaide), 19 Fullarton Road, Fullarton.

Hicks, Neil Dennis, M.B., B.S., 1948 (Univ. Adelaide), 11 Grandview Grove, Toorak Gardens.

Medical Prizes.

DAVID ANDERSON-BERRY PRIZE (1950).

A DAVID ANDERSON-BERRY SILVER-GILT MEDAL, together with a sum of money amounting to about £100, will be awarded during 1950 by the Royal Society of Edinburgh to the person who, in the opinion of the Council, has recently produced the best work on the therapeutical effect of X rays on human diseases. Applications for this prize are invited. They may be based on both published and unpublished work and should be accompanied by copies of the relevant papers. Applications must be in the hands of the General Secretary, Royal Society of Edinburgh, 22 George Street, Edinburgh, 2, not later than March 31, 1950.

Notice.

INTERNATIONAL EXHIBITION OF MEDICAL PHOTOGRAPHY.

THE Medical Group of the Royal Photographic Society will be holding their annual exhibition early in the summer of 1950, and invite all medical photographers to submit their work for selection. The committee of the Group feel that the time is now opportune for making their annual exhibition international and that great benefit will accrue both to the photographers and to the science of medical photography.

For the exhibition both still and cinematograph pictures in monochrome or colour are invited. Prints from abroad may be unmounted; these will afterwards be put onto standard 16 inch by 20 inch and 12 inch by 15 inch mounts. The necessary descriptive matter must be sent with the prints. Entry forms may be obtained from Kenneth G. Moreman, Honorary Secretary, Medical Group, 16 Princes Gate, London, S.W.7.

AUSTRALIAN RED CROSS SOCIETY.

THE National Council of the Australian Red Cross Society, through its National Blood Transfusion Committee, has arranged a scientific meeting on blood transfusion which will be held at 8.15 p.m. on Thursday, February 23, 1950, in the lecture theatre at the Royal Melbourne Hospital, Melbourne.

The programme will be as follows: "Survey of the Incidence and Effects of Rh Incompatibility", Laboratory Features, Dr. L. M. Bryce and Dr. R. Jakobowicz; Clinical Features, Dr. K. Campbell. "Some Aspects of the Management of Hæmatemesis", Dr. P. J. Parsons. "Miracle Fluid", "Technicolour" film presented to the Australian Red Cross Society by the Canadian Red Cross Society.

All members of the British Medical Association are cordially invited to attend the meeting and to take part in the discussion following the papers.

Medical Appointments.

Dr. M. C. Fowler has been appointed medical bacteriologist at the Institute of Medical and Veterinary Science, Adelaide.

Diary for the Month.

FEB. 13.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
FEB. 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 16.—Victorian Branch, B.M.A.: Executive Meeting.
FEB. 21.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 22.—Victorian Branch, B.M.A.: Council Meeting.
FEB. 23.—South Australian Branch, B.M.A.: Clinical Meeting.
FEB. 24.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 28.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135 Macquarie Street, Sydney): Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester United Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £3 *per annum* within Australia and the British Commonwealth of Nations, and £4 10s. *per annum* within America and foreign countries, payable in advance.